THE INFLUENCE OF EATING DISORDERS ON FERTILITY TRAJECTORIES

by

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ABSTRACT

There is a well-documented link between eating disorders and adverse physical health outcomes, infertility in particular. Fertility marks an important transition to adulthood, setting the stage for future opportunities or experiences. Drawing on sociological theories of the life course, this study explores the ways in which eating disorders influence fertility trajectories of women. Using data from the National Longitudinal Study of Adolescent Health and the Utah Population Database, this study offers innovative and substantial contributions to the current literature on eating disorders and fertility by (1) assessing how sampling and eating disorder measurement shapes the inferences we make about the influence of eating disorders on fertility, (2) addressing potential familial and genetic confounding factors by utilizing a sibling-comparison design, (3) assessing the influence of eating disorder disease type on fertility outcomes, and (4) proposing and testing potential social mechanisms through which eating disorders influence fertility outcomes. The results indicate that the influence of eating disorders on the parity and fertility timing of women is complex. Sampling and measurement shapes the inferences we make about the influence of eating disorder on fertility outcomes, and fertility trajectories vary by eating disorder disease type. Theoretical and methodological explanations for these results are discussed as well as future research directions.

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CHAPTER 1

EATING DISORDERS AND THE LIFE COURSE: RESEARCH QUESTIONS AND THEORETICAL BACKGROUND

Eating Disorders and Disordered Eating Behavior: An Introduction

Eating disorders involve a set of severe negative attitudes and behaviors surrounding weight, physical appearance, and food (Fairburn and Harrison 2003). Today, there are four diagnosable eating disorder categories currently recognized in medical manuals (i.e., the ICD-10 and DSM-5) and include: (1) anorexia nervosa (AN), characterized by excessive low body weight and an obsessive fear of gaining weight; (2) bulimia nervosa (BN), characterized by recurrent binge eating followed be compensatory behaviors such as purging, use of laxatives/diuretics, or excessive exercise; (3) binge eating disorder (BED), characterized by binge eating at least 2-3 times a week along with feelings of shame or guilt after overeating; (4) other specified feeding or eating disorder (OSFED), which is an eating or feeding disorder that does not meet full DSM-5 criteria for AN, BN, or BED (American Psychiatric Association [APA] 2013). This category includes, for example, individuals who may exhibit AN criteria, but are not underweight, or individuals who purge without binging (APA 2013).

More recently, eating disorder research has focused on disordered eating behaviors, which are the behavioral symptoms of eating disorders. Examples of disordered eating behaviors include purging, binge eating, and nonpurging compensatory

behaviors (e.g., fasting or taking laxatives). Survey research illustrates that engaging in disordered eating behaviors is much more common than having a diagnosed eating disorder (Stephen et al. 2014; Tabler and Utz 2015). Disordered eating behaviors have become increasingly important to the study of eating disorders, as they have been shown to have clinical and conceptual relevance (Stephen et al. 2014). For example, studies have shown that vomiting or misusing laxatives or diuretics to manage weight (even occasionally) predicts excess weight gain, and is associated with low self esteem, substance use, and impaired psychosocial adjustment (Keel and Striegel-Moore 2009; Piran and Robinson 2011).

While it may be a commonly held view that eating disorders are an individual's lifestyle choice (Crisp 2005), both eating disorders and disordered eating behaviors represent a serious and often fatal chronic mental illness that disproportionately affects the health and wellbeing of women (Polivy and Herman 2002). Eating disorders have one of the highest estimated mortality rates (4-6 percent) of any mental disorder (Crow et al. 2009; Sullivan 1995), are predictive of suicide (Chesney, Goodwin, and Fazel 2014; Preti et al. 2011; Rosling et al. 2011), and are associated with additional comorbid mental health conditions including depression, and substance abuse (Krahn 1991; Piran and Robinson 2011; Santos, Richards, and Bleckley 2007). Eating disorders are the third most prevalent chronic condition affecting adolescent females, behind obesity and asthma (Fisher et al. 1995; Lucas et al. 1991). Eating disorders contribute significantly to the economic and social burden of disease in the United States (Crow and Peterson 2003) and globally (Simon, Schmidt, and Pilling 2005; Whiteford et al. 2013).

Although eating disorders are highly medicalized, that is, they are problems that

have come to be defined and treated as legitimate medical conditions (Conrad 1992), eating disorders are highly stigmatizing; prior surveys of public opinions in 1998 and 2004 found that the public was likely to perceive eating disorders negatively, and were likely to identify eating disorders as self-inflicted and egocentric (Crisp 2005). Recent research suggests that mainstream attitudes toward individuals with eating disorders are significantly more stigmatizing than attitudes towards individuals with depression (Roehrig and McLean 2010). The potentially stigmatizing nature of eating disorders may result in additional social and psychological burdens to individuals who suffer from them, and ultimately restrict opportunities for their treatment (Gowers and Shore 1998); while upwards of 24 million individuals in the US suffer from an eating disorder, only 1 in 10 men and women are estimated to receive treatment (Noordenbos et al. 2002).

Potentially due to their stigmatizing nature, evidence suggests that eating disorders result in poor social adjustment in adolescence and young adulthood, including higher rates of delinquent behavior, substance abuse, and unemployment (Stephen et al. 2014; Striegel-Moore, Seeley, and Lewinsohn 2003). In a recently completed study, Tabler and Utz (2015) found that adolescents or young adults with eating disorders, especially females, experienced reduced levels of socioeconomic achievement in early adulthood.

Eating disorders influence the physical, mental, and social wellbeing of those who suffer from them, and may act as a turning point that sets individuals on a trajectory of disadvantage, whereby they do not have the same opportunities for success. The focus of this dissertation is to bridge epidemiological, psychological, and sociological research on eating disorders in order to better assess whether eating disorders act as a turning point

that disrupts successful adult development. In particular, this study seeks to expand our understanding of eating disorders by assessing how eating disorders influence parenthood and fertility experiences, using both community and clinical samples.

Below, I outline the life course framework I utilize within this dissertation, highlighting how life course concepts and principals can help us develop a better understanding of eating disorders. Later, I embed current eating disorder research within a life course context in order to bridge disciplinary divides and illustrate the unique contributions epidemiological, psychological, and sociological research have contributed to what we know about the early life etiology, the diagnosis and treatment of eating disorders in adolescence and young adulthood, and the later life consequences of eating disorders.

Defining Eating Disorders within a Life Course Framework

Eating disorders and related behaviors should be considered from the perspective of the individual's life course, that is, eating disorder research should take greater consideration of the individual's life path or developmental trajectory. The life course perspective emphasizes that early life context influences outcomes in later life by setting the individual on a particular life trajectory (Mortimer and Shanahan 2003). Very little research has examined eating disorders across the individual's lifespan, from birth or early adolescence, through adulthood or until death. Instead, most eating disorder research relies heavily on cross-sectional or short duration research. Some longitudinal studies of eating disorders are available, although they have focused primarily on etiology and health-related outcomes (see for example Austin et al. (2009)).

Life course theory also offers a set of concepts that could be applied or used to

increase our understanding of eating disorders, such as a "turning point." A turning point is defined as a particular event, experience, or set of behaviors that result in a significant change to the life or health trajectory of an individual (Wheaton and Gotlib 1997). An individual's life path is rarely straight, influenced by historical events, individual experiences and choices, and cultural or normative expectations. Those events that result in a significant change to the life trajectory of individuals are referred to as "turning points" (Wheaton and Gotlib 1997). Turning points are not necessarily dramatic events, or even a single discrete event (Wheaton and Gotlib 1997). Rather, turning points can be a set of behaviors that result in a gradual, but significant change in the direction of an individual's life trajectory. This concept is illustrated in Figure 1.1. The theoretical concept of a turning point may apply to eating disorders, which involve a set of negative health behaviors tied to feelings of negative self-image.

The transition to adulthood can be a highly complex process; most adolescents have been dependent on parents but with age are transitioning into independence both financially and emotionally (Jekielek and Brown 2005). Some adolescents experience setbacks including premature parenthood, dropping out of high school, and failing to find a job (Jekielek and Brown 2005). These setbacks place the young adult on a disadvantaged trajectory, leading to cumulative disadvantage in socioeconomic and emotional stability later in life (Jekielek and Brown 2005). Cumulative disadvantage theory argues that turning points in early life can have cumulative negative effects, setting the individual on a trajectory of even more disadvantage later in life (O'Rand 1996; Ferraro and Kelley-Moore 2003; Wadsworth 1997; Wadsworth 1991). Similarly, the presence of eating disorders during adolescence may set a child on to a different path,

resulting in differential successes during the transition to adulthood and into the later stages of adulthood.

It is well established that eating disorders result in unfavorable physical and mental health outcomes that persist well into adulthood (Berkman, Lohr, and Bulik 2007). Preliminary evidence suggests that eating disorders result in poor social adjustment in adolescence and adulthood, including delinquency, higher unemployment, and lower levels of academic achievement (Stephen et al. 2014; Striegel-Moore et al. 2003; Tabler and Utz 2015). These results suggest that eating disorders may act as a turning point that sets individuals on a trajectory of disadvantage, whereby they do not have the same opportunities for success.

Eating disorders should therefore be re-examined from a life course perspective, where individuals experience early life risks for eating disorders in childhood, are likely to develop an eating disorder in early adolescence, be diagnosed sometime in late adolescence or young adulthood, and experience negative consequences for physical, emotional, and social wellbeing in early adulthood. In the next several paragraphs, I will discuss research pertaining to the prevalence and early life etiology of eating disorders, the diagnosis and treatment of eating disorders in adolescence and young adulthood, and the later life consequences of eating disorders.

Prevalence and Early Life Etiology of Eating Disorders

Prevalence and Manifestation

Swanson et al. (2011) estimate that approximately 2.7 per 100,000 adolescents between the ages of 13-18 in the United States have an eating disorder. Since many eating disorders are not diagnosed, these estimates are likely underrepresented measures

of true prevalence. Eating disorders often begin in adolescence, with an average age of diagnosis at 17-18 years (Fairburn and Harrison 2003). There is also evidence that the risk factors for, and early symptoms of, eating disorders develop in elementary and middle school years (ages 10-13) (White 2000). Longitudinal studies indicate that disordered eating behaviors increase from early to late adolescence, meaning an adolescent's risk for developing and engaging in eating disorders or disordered eating behaviors increases across adolescence (Neumark-Sztainer et al. 2006).

Given that adolescence is an important marker of human development where increased choice and autonomy are often expressed (Steinberg and Silverberg 1986), it is not surprising that this is the life stage in which most eating disorders and related behaviors are first noticed. Eating disorders are often chronic, persisting well into adulthood, with full recovery only occurring for approximately half of adolescents who actually receive treatment (Yager and Andersen 2005). Because eating disorders and disordered eating behaviors manifest in adolescence and have a high likelihood of becoming chronic lifelong conditions, longitudinal methodologies that embed eating disorders within the life course are arguably fitting and necessary.

Etiological Factors

Genetics, Environment, and Socio-demographic Characteristics

Figure 1.2 illustrates the etiology of eating disorders. Etiological factors for eating disorders often occur in childhood; however, current evidence suggests that the etiology of eating disorders may be the result of genetic predisposition, although identifying specific genes has proved difficult, given that there is strong evidence that the heritable traits of eating disorders have a "polygenetic," or additive genetic, pattern of inheritance.

That is, several genes are likely responsible for the development of the eating disorder "trait" (Trace et al. 2013). Evidence suggests that eating disorders are the result of epigenetics, that is, the interaction between the environment and genes (Campbell et al. 2011; Goodman et al. 2014; Strober et al. 2000). When individuals' genetic predispositions interact with environments rich in potential risk factors, such as a stressful home environment, their likelihood of developing an eating disorder or disordered eating behavior increases.

In addition, there is often a high risk of familial transference associated with eating disorders, where families "pass" eating disorders through the family environment from parent to child (Polivy and Herman 2002; Strober et al. 2000). For example, mothers with eating disorders have higher expectations for their child's thinness, may constrict the diets of their child, and are less positive about their child's "attractiveness," prompting the child to engage in disordered eating behaviors (Polivy and Herman 2002). The home environment has an important influence on the development of eating disorders and/or disordered eating behaviors. In a systematic review of the causes of eating disorders, Polivy and Herman (2002) identify negative family environments and lack of familial support during early childhood as major risk factors for eating disorders. Eating disorders are often understood as coping mechanisms for the perceived lack of control or support in the home environment (Polivy and Herman 2002; Wagener and Much 2010).

Also, the larger social and peer environment is important to the etiology of eating disorders. Many modern societies emphasize culturally idealized thinness, and individuals with eating disorders are aware of the importance of this ideal, internalize it,

and perceive pressure from the media and their peers to be thin (Levine and Murnen 2009). Although researchers have grappled over how we might change predominating cultural perspectives idealizing thinness (Levine and Murnen 2009), there is evidence that increased positive social support from peers and other adults is protective against disordered eating and the development of eating disorders, thereby mitigating some of these unhealthy cultural pressures (Limbert 2010; McVey et al. 2003).

Existing research indicates that there are socioeconomic status (SES), racial, and ethnic differences in type-specific eating disorder symptomology and prevalence (Franko et al. 2007; Swanson et al. 2011). Whites and individuals from higher SES backgrounds experience higher prevalence of anorexia nervosa (Swanson et al. 2011), while African Americans and Latinos are found to have higher prevalence of bulimia nervosa (Marques et al. 2011). Racial, ethnic, and SES differences in eating disorder prevalence may be related to racial and ethnic differences in perceptions of body weight and what constitutes an "ideal body"; for example, compared to black women, white women consider themselves to be larger and report a lower ideal bodyweight (Stevens, Kumanyika, and Keil 1994).

Gender and Sexual Orientation

The vast majority of research on eating disorders and disordered eating behaviors have focused on female samples, although there is a growing literature on eating disorders and disordered eating behaviors among men. Females have been consistently shown to have higher lifetime prevalence of eating disorders than males, and are more likely to engage in disordered eating behaviors (Hudson et al. 2007; Santos et al. 2007; Stephen et al. 2014). Yet many studies indicate clinical similarities between males and

females with eating disorders (Woodside et al. 2001), and that males and females have similar patterns of age at onset (Woodside et al. 2001), but different symptomology (Lewinsohn et al. 2002). Previous studies also suggest that the etiological factors influencing eating disorders or disordered eating behavior development is similar for both males and females (Burns and Crisp 1985; Gueguen et al. 2012; Polivy and Herman 2002; Woodside et al. 2001).

In addition, sexual minority status (i.e., identifying as gay or bisexual) is a clear risk factor for an eating disorder among men (Carlat, Camargo, and Herzog 1997; Feldman and Meyer 2007). Researchers posit that, like heterosexual women, gay men are dissatisfied with their bodies and vulnerable to eating disorders because of a shared emphasis on physical attractiveness and thinness that is based on a desire to attract and please men. There is some evidence to support this hypothesis; lesbian and heterosexual men have been found to be less concerned with their own physical attractiveness, and would hypothetically then be less dissatisfied with their bodies and less vulnerable to eating disorders (Siever 1994; Strong et al. 2000).

However, the empirical literature is mixed regarding whether or not sexual minority status is a risk factor for eating disorders among women; for example, the rate of bulimia nervosa and binge eating is similar if not higher among sexual minority women (including bisexual or lesbian) compared to heterosexual women (Austin et al. 2009; Heffernan 1996), while other research has failed to detect any eating disorder differences among these groups (Feldman and Meyer 2007). It is also important to note that there have been subgroup differences detected among sexual minorities. For example “mostly heterosexuals” and “bisexuals,” were found to have higher levels of purging/bulimia

while “lesbian” women were only found to have higher rates of binge eating disorder or behaviors (Austin et al. 2009; Heffernan 1996). Therefore, it is important to consider sexual minority status, and even within sexual minority status group differences, when exploring eating disorder etiology and outcomes.

Although eating disorders and disordered eating behaviors are the result of a myriad of risk factors and complex genetic and epigenetic processes. As illustrated in Figure 1.2, it is suggested that the early life context of a child—be it their socio-demographic resources or the social support they receive—shapes his or her risk for eating disorders or disordered eating behaviors. Therefore, high-quality eating disorder research will consider or adjust for underlying etiological factors, such as race/ethnicity, social support in childhood, sexual orientation, gender, and childhood SES, within their models.

Diagnosis and Treatment of Eating Disorders

Eating disorders are notoriously under diagnosed, with very few individuals seeking treatment from a healthcare professional, and an even smaller minority receiving appropriate mental health care (Hart et al. 2011). In a systematic review of the treatment literature, Hart et al. (2011) found that individuals from community samples with a diagnosable eating disorder were more likely to seek help for weight loss than direct treatment for an eating problem. However, for those who do receive treatment, they generally begin to receive treatment in late adolescence or young adulthood (between ages 18-24) (Lock and le Grange 2005; Yager and Andersen 2005).

Many treatment studies lack necessary statistical power, randomization, and robustness to yield meaningful results (Lock and le Grange 2005; Van den Eynde and

Schmidt 2008). However, when psychotherapy and/or pharmacotherapy strategies are utilized, there is some evidence that treatment can be moderately effective, particularly when the intervention occurs in adolescence rather than adulthood (Lock and le Grange 2005; Yager and Andersen 2005). Mindfulness-eating awareness intervention, where individuals are taught to cultivate self-acceptance, meditation, and emotional regulation, is an emerging form of eating disorder psychotherapy that has been found to be promising and empirically supported (Kristeller and Wolever 2010; Wanden-Berghe, Sanz-Valero, and Wanden-Berghe 2010). Eating disorder treatment strategies that involve the parents of adolescents have been found to be particularly beneficial for long-term recovery (Lock and le Grange 2005). Conversely, treatment in adulthood consistently yields poor results, with greater likelihood of short-term recovery and relapse (Lock and le Grange 2005; Yager and Andersen 2005).

In short, most individuals, including those who receive some form of appropriate therapy, are likely to experience eating disorders as chronic conditions. In addition, there is overwhelming consensus that receiving treatment, whatever form it may take, is most beneficial when it occurs in adolescence. It is therefore important to consider when an individual receives treatment, as well as the treatment form, when examining the long- and short-term consequences of eating disorders.

Consequences of Eating Disorders

Physical and Mental Health

Eating disorders are known to result in deleterious health. The negative consequences of eating disorders are illustrated in Figure 1.3. Negative physical health symptoms such as heart arrhythmias and renal failure have been observed in individuals

as young as 15 (Nicholls, Lynn, and Viner 2011). Certain forms of disordered eating behaviors in adolescence, such as purging and binging, have been linked to increases in BMI in adulthood (Stephen et al. 2014). The health consequences of eating disorders, such as the effects of chronic malnourishment, may have lasting effects throughout the life course even after the behaviors have ceased (Johnson et al. 2002). The physical health consequences of eating disorders, such as osteoporosis and anemia, are similar for men and women (Norris et al. 2012). It is commonly suggested that the more severe and prolonged eating disorders have the longest lasting effects on physical health (Yager and Andersen 2005).

In addition, mental health is known to influence and be influenced by eating disorders and disordered eating behaviors as illustrated by the bi-directional arrow linking mental health and eating disorders in Figure 1.3; mental health comorbidities are common among individuals with eating disorders (Santos et al. 2007; Swanson et al. 2011). Individuals with eating disorders often experience high levels of stress and depression, as well as a myriad of other mental and emotional health comorbid conditions, such as post-traumatic stress disorder (PTSD) and substance abuse (Norris et al. 2012; Swanson et al. 2011). There is evidence that eating disorders perpetuate poor mental health outcomes (Berkman et al. 2007). Like physical health, there are gender similarities in the mental health outcomes of eating disorders; males and females with eating disorders are both likely to exhibit depression and anxiety (Norris et al. 2012; Stephen et al. 2014; Woodside et al. 2001).

Social Wellbeing

Eating disorders have drastic and long-lasting influences on the mental, physical, and health-related quality of life of individuals who suffer from them. Therefore, it is important to consider how eating disorders may be influencing other aspects of the individual's life, such as parenthood, and how the health consequences of eating disorders may be acting as a mechanism through which eating disorders influence adult development. The health consequences of eating disorders have large implications for the quality of lives of those who suffer from them. Health-related quality of life (HRQOL) conceptualizes wellbeing as not just "health" but psychological, social, and physical wellbeing (Engel et al. 2009). Compared with the general female population, aged 18-34 years, subjects with eating disorders are shown to have global deterioration in their perception of their HRQOL (Padierna et al. 2000). Padierna et al. (2000) note that psychosocial deterioration was most evident in the areas of vitality, emotional role, social functioning, and mental health (Padierna et al. 2000).

In addition, a much smaller body of research has shown that eating disorders or disordered eating behaviors may be disruptive to adolescent's successful transition to adulthood. For example, individuals with eating disorders are at risk for greater delinquency in adolescence and experience higher rates of unemployment, and report low life satisfaction, in adulthood (Stephen et al. 2014; Striegel-Moore et al. 2003). Eating disorders and disordered eating behaviors negatively influence socioeconomic independence in early adulthood, particularly for women (Tabler and Utz 2015); females with eating disorders or disordered eating behaviors in adolescence were found to earn less personal income, achieve less years of education, and have lower likelihood of

owning a home, in early adulthood (Tabler and Utz 2015). These findings illustrate that individuals with eating disorders may experience a disruption in their ability to achieve socioeconomic independence and other markers of social wellbeing in early adulthood.

It is important to note that the disruption to the transition to adulthood is likely both a direct and indirect result of eating disorders. Preliminary evidence suggests that eating disorders have both a direct effect on socioeconomic achievement and family formation in adulthood, but also an indirect effect, influencing family formation through resulting physical and mental health outcomes (Tabler and Utz 2015). These indirect and direct effects are illustrated in Figure 1.3 where both the eating disorder and resulting physical and health outcomes are noted to influence family formation.

Childbearing and Fertility

In addition, an individual's physical and mental health is known to influence, and be influenced by, childbearing, with both biological and social mechanisms playing a role. It is important to note that most fertility studies sample women with only anorexia and/or women who are trying to get pregnant, and may therefore be unrepresentative of the overall fertility experience of individuals with eating disorders. Additional research should be done to expand our understanding of the fertility experiences of individuals with eating disorders by assessing birth timing (e.g., age at first birth) and parity (e.g., number of children). Understanding the overall fertility experiences of individuals with eating disorders will provide better insight into their potential long-term consequences.

While the etiology and physical and mental health consequences are similar for males and females, it is important not to assume that eating disorders will have similar influences on fertility trajectories of men and women. Although the physical

manifestation and physical outcomes related to eating disorders may be similar for men and women (which may be due to the fact that many studies are engaging in a comparison of sex or sex category differences), it is important to keep in mind that gender is a *social* construct, and therefore *social* outcomes may vary by gender. When men and women appear to function similarly in relation to a given phenomenon, we sometimes have the tendency to minimize differences, that is, engage in "beta bias," where the similarity or equality between men and women may become over stressed (Hare-Mustin and Marecek 1988). It is important to consider our own potential to engage in "beta bias." Although it is important to consider potential gender differences when assessing the relationship between eating disorders and markers of adult development, this study focuses specifically on women, given that there are complex social and physical aspects to fertility that differ for men compared to women.

Health Mechanisms

There are two different approaches to examining the link eating disorders and disordered eating behaviors and fertility outcomes. Clinical studies indicate that women with prolonged or severe eating disorders may have difficulty getting pregnant as a result of physical health complications (James 2001; Linna et al. 2013; Stewart et al. 1990). As noted, it is well established that eating disorders and disordered eating behaviors are associated with long-lasting mental and physical health consequences (Berkman et al. 2007; Hudson et al. 2007; Nicholls et al. 2011; Norris et al. 2012; Swanson et al. 2011). More severe and prolonged eating disorders and disordered eating behaviors have the most severe and longest-lasting effects on physical and mental health (Yager and Andersen 2005).

The physical health consequences of eating disorders, weight-related menstruation disruption in particular, have been linked to lifetime fertility complications (Freizinger et al. 2010), with women with eating disorders expressing difficulty becoming pregnant and/or experiencing longer times to conception (Easter, Treasure, and Micali 2011). In addition, major depression has been found to have a direct influence on ovarian function, which may result in reduced or disrupted fertility (Harlow et al. 2003). There is evidence to conclude that eating disorders may reduce overall parity, or number of children born; Linna et al. (2013) found that women seeking treatment for eating disorders were more likely to be childless than the control group in an observational study utilizing a clinical sample. Based on this body of medical research, we might hypothesize that women with adolescent eating disorders or disordered eating behaviors will have fewer children in early adulthood.

However, there is an aberrant finding within medical studies of eating disorders and fertility that may indicate that women with eating disorders or disordered eating behaviors may actually be at risk of having a child at an early age, or multiple children in early adulthood; recent research indicates that women with eating disorders are at greater risk of experiencing unplanned pregnancy, particularly those studies utilizing broader community samples (Bulik et al. 2010; Easter et al. 2011). Unplanned pregnancy may be indicative of early entry into parenthood and high overall parity. In the next section, I propose an alternative set of social mechanisms that may provide insight into recent findings linking eating disorders and unplanned pregnancy, and suggest a counter-hypothesis that eating disorders and disordered eating behaviors may actually be associated with higher parity in early adulthood.

Social Mechanisms

Sociological research suggests an alternative understanding of the link between eating disorders or disordered eating behaviors and early adult fertility. Disordered eating behaviors have been conceptualized as a form of internally directed deviance, resulting from negative self-feelings (Sischo, Taylor, and Yancey Martin 2006). In addition, eating disorders and disordered eating behaviors have been associated with forms of externalized deviance; for example, eating disorders and disordered eating behaviors in adolescence are also associated with higher levels of delinquency and substance use (Piran and Robinson 2011; Stephen et al. 2014; Striegel-Moore et al. 2003). Such risky behaviors are also commonly associated with risky sexual behavior, including early age of first sexual encounter and higher numbers of sexual partners, and early or unintended pregnancy (Naimi et al. 2003; Pugh et al. 1990; Yamaguchi and Kandel 1987).

Furthermore, research indicates that young adults diagnosed with one or more psychiatric disorders, including depression, eating disorders, anxiety, and antisocial disorder, are more likely to engage in risky sexual intercourse (noncondom use, higher number of partners) and have sexual intercourse at an early age (Ramrakha et al. 2000; Shrier et al. 2001). Young women with eating disorder or disordered eating behavior may be particularly prone to risky sexual behavior, including earlier ages of first sexual intercourse and higher number of sexual partners, due to their comprised self-esteem (Fisher et al. 1991). In addition women with eating disorders or disordered eating behavior may be less likely to use effective forms of contraception due to their assumption that they may be infertile due to their compromised health status (Bulik et al. 2010; Downs et al. 2004), resulting in higher rates of unplanned pregnancy (Bulik et al.

2010; Easter et al. 2011).

Because unintended pregnancy is a risk factor for subsequent, unintended pregnancy (Kuroki et al. 2008), women with eating disorders or disordered eating behavior may not only be at risk of early entry into parenthood, but of experiencing multiple births at a younger age than their unafflicted peers. This study builds on research that has shown that women with eating disorders are at greater risk of experiencing unplanned pregnancy, particularly those studies utilizing broader community samples (Bulik et al. 2010; Easter et al. 2011). Based on the sociological literature, researchers may expect eating disorders and disordered eating behaviors to be associated with earlier age at first birth, and higher overall parity.

More robust research can be done to improve our understanding of how eating disorders shape family formation in general, and the transition to adulthood in particular. Understanding the adult development of individuals with eating disorders across their life course—their fertility behaviors in particular—would provide insight into the mechanisms through which those individuals may be accruing poor physical and mental health.

Eating Disorders Across the Life Course

Figure 1.4 illustrates eating disorders from a life course perspective. The line of Figure 1.4 directly suggesting a link between eating disorders and childbearing can perhaps be explained by the notions of cumulative disadvantage theory whereby eating disorders can be conceptualized as a potential turning point that sets an individual on to a different trajectory in terms of social wellbeing in adulthood. However, the theoretical model in Figure 1.4 also acknowledges that both the etiological factors that shape eating

disorders, *and* the subsequent pathology that follows eating disorders, will have negative effects on the individual's transition to adulthood. For example, the opportunities and constraints associated with early life contexts—such as having an unsupportive family or peer network—may also have a direct influence on one's social wellbeing, due to lack of social and financial resources (Caspi et al. 1998; Ramrakha et al. 2000). Indeed, it is well documented that low socioeconomic status in early life constrains opportunities and leads to poorer socioeconomic attainment and higher rates of unplanned pregnancy in adulthood (Duncan et al. 1998; Rindfuss, St. John, and Bumpass 1984; Sirin 2005).

Similarly, the poor mental or physical health associated with eating disorders likely shape social wellbeing. For example, poor physical and mental health are known to disrupt one's ability to pursue education or be successful in a workplace, lower his or her likelihood of a successful marriage, and contribute to fertility issues (Kessler et al. 1995; Lorant et al. 2003; Maslow et al. 2011; Wang et al. 2014). In addition, lower level of education (which may result from poor health) is a known risk factor for early ages at first birth and unplanned pregnancy (Rindfuss et al. 1984). Eating disorders often result in poor physical and mental health, yet poor physical and mental health has an influence on social wellbeing. Therefore, physical and mental health conditions in adulthood are likely mechanisms through which eating disorders disrupt adult development. Thus, it is important to consider how eating disorders during adolescence may have both direct and indirect effects on the transition to adulthood.

Only by viewing eating disorders from a life course perspective can we see how eating disorders have the potential to influence, either directly or indirectly, other domains of life, such as the transition to parenthood. Given the notions of cumulative

disadvantage, turning points, and life course trajectories, it is important for future research exploring the influence of eating disorders on adult development to consider the confounding influences of early life contexts, genetic predisposition, as well as the immediate health-related outcomes of eating disorders. Such research is important theoretically because it attempts to understand the validity of life course theory and life course concepts as they apply to eating disorders. It is important practically because it has the potential to illustrate how eating disorders disrupt, not only adolescent physical and mental health, but also their capacity to appropriate new roles and statuses of adulthood.

Throughout these chapters, I seek to approach eating disorders from a life course perspective, embedding findings pertinent to etiology and outcomes research within this framework, while utilizing nationally representative and community based samples. By so doing, I am able to illustrate the potential gaps in our understanding of how eating disorders may be affecting those who suffer from them. Utilizing large, longitudinal, population- and/or community-based samples to assess the influence of eating disorders on the transition to adulthood is an important next step in eating disorder research. The present study, which seeks to compare the longitudinal information available in the National Longitudinal Study of Adolescent and Young Adult Health (Add Health) and that available through the Utah Population Data Base (UPDB), has three primary aims.

Study Objectives, Data Sources, and Samples

Study Objectives

The first objective is to assess the influence of eating disorders on fertility trajectories, and illustrate how measurement influences the results of eating disorder research; in Chapter 2, I compare the age at first birth and parity of women who have

been hospitalized or treated for an eating disorder in the State of Utah to that of a nationally representative sample of young women who self-identify as having been diagnosed with an eating disorder or engage in disordered eating behaviors.

The second objective is to address the role of familial and shared early life environment on fertility trajectories of women with eating disorders, and examine how eating disorder type and severity influences fertility trajectories; in Chapter 3, using data from the Utah Population Database, I compare the age at first birth and parity of women in early adulthood who have been diagnosed with an eating disorder, to that of their closest aged sister who has never been diagnosed with an eating disorder. Although the literature suggests that eating disorders are rooted in familial and shared early life environmental, and/or epigenetic etiology (Campbell et al. 2011), very few studies have actually addresses these potential confounding factors in outcomes research. In addition, I more closely examine how eating disorder type (anorexia nervosa, bulimia nervosa, or eating disorder-NOS) influences fertility trajectories when accounting for measures of disease severity and treatment timing.

The third objective is to suggest and test potential social mechanisms through which eating disorders may influence fertility, namely deviance and sexual risk-taking; In Chapter 4, using data from the National Study of Adolescent Health, I examine more closely adolescent delinquency and sexual risk-taking as potential explanatory mechanisms through which eating disorders influence female fertility. In this chapter, I examine the influence of eating disorders or disordered eating behaviors on parity (number of children) born to women in early adulthood. These chapters act as a counterpoint to traditional biomedical literature, which suggests eating disorders result in

delayed fertility and fertility complications for women.

Finally, Chapter 5 summarizes the findings of the previous three chapters, noting the implications and limitations of each. One limitation in particular that will be revisited both in Chapter 5 and periodically throughout the project is that the findings reported here are bound by measurement. In other words, the extent to which such findings apply to different types of samples with different eating disorder or disordered eating behavior measurements is unknown. Finally, potential directions for future research on eating disorders and family formation outcomes are discussed.

Data Sources

To address the *first study objective,* which is to assess the influence of eating disorders on fertility trajectories, and how measurement and sampling may influence this relationship, I will use two data sources: the Longitudinal Study of Adolescent and Young Adult Health (Add Health) and the Utah Population Database (UPDB). To address the *second study objective,* which is to assess the extent to which shared early life environment and familial relationships explain the relationship between eating disorders and fertility, I will rely only on the UPDB data. To address the *third study objective,* to test potential social mechanisms through which eating disorders influence fertility trajectories, I will use Add Health only. Descriptions of these data sources are as follows.

Add Health

The present study utilizes data from the restricted-use (full sample) National Longitudinal Study of Adolescent Health (Add Health) survey Waves I (1994–1995), II (1996), III (2001–2002), and IV (2008-2009). Add Health has been following over

20,000 American adolescents over the course of 14 years, with four waves of data collection since the study's inception when participants were in grades 7–12. Wave I was conducted between 1994 and 1995, with sampling from 132 schools in the United States, representative for region, urbanicity, size, and ethnicity. Initially, 80 high schools were recruited, and each participating school identified a feeder middle school in the community, with the requirement of the inclusion of 7th graders. A total of 20,074 adolescents in grades 7–12 were tested using self-administered, in-school questionnaires. Wave II was conducted in 1996, and included over 15,000 of the participants originally tested in Wave I, and excluded those who were no longer in school. In-home interviews were conducted with available original Wave I participants at Wave III and Wave IV. Further details about the study design can be found elsewhere (Harris 2011).

The Utah Population Database

The present study also uses data from the Utah Population Database (UPDB). The UPDB is limited to the Utah population, and contains socio-demographic, family, medical, and vital records linked into large multigenerational pedigrees. The UPDB contains information on nearly 8 million individuals. UPDB staff links vital records and multigenerational pedigrees to medical records provided by the University of Utah Hospitals and Clinics (UUHC) and hospitalization records provided by the Utah Department of Health (UDOH) and height, weight, and residence information provided by the Department of Motor Vehicles (DMV). It is annually updated with records of Utah birth and death certificates, driver licenses, and extensive medical and vital records. The Utah Resource for Genetic and Epidemiologic Research (RGE) administers access to these data through a review process of the project proposal, and all research requires

human subjects and RGE approval (Wylie and Mineau 2003). The confidentiality of individuals represented in these records is maintained based on agreements between RGE and the data contributors.

Large population-based data sets that are able to identify familial patterning of eating disorders are key to understanding whether genetics play a role in the physical and mental health outcomes of eating disorders. Few studies have been able to address the potential genetic and epigenetic confounding factors that may play a part in how early life social conditions shape health. The pedigree (family) linkages in the UPDB allows for the use of a sibling-comparison design, which in turn allows the research to address some potential confounding genetic and early life factors. There are very few available data sources that have the high-quality medical, vital, socio-demographic, pedigree data necessary for this type of study design.

Samples

Study Objective 1: Assessing the Influence of Eating Disorders on Fertility Trajectories

For this aim, I will utilize samples from both Add Health and the UPDB, which compare persons with eating disorders to those without eating disorders. The sample from Add Health includes individuals who have been diagnosed with an eating disorder or engage in eating disorder behaviors (n=1,430), and a group of peers without an eating disorder or related behavior (n=4,522). This results in a total analytic sample of 5,952 individuals. The UPDB sample includes 1,725 individuals who were treated for an eating disorder between 12 and 32, and a general population comparison sample of 1,725 individuals captured in the UPDB have been treated for an eating disorder between the

ages of 12 and 32. These individuals are compared to randomly generated sample of 10,396 women who have never been treated for an eating disorder, who are not biologically related.

Study Objective 2: Addressing the Role of Familial and Shared Early Life Environment, and Exploring Differences in Fertility by Eating Disorder Type

For this aim, I utilize UPDB data only. This study will include three analytic samples: a sibling sample, a general population sample, and an eating disorder sample. The eating disorder analytic sample includes 1,565 women, 392 women diagnosed with anorexia, 442 women diagnosed with bulimia, and 731 diagnosed with eating disorder, not otherwise specified. The sibling sample includes 506 women diagnosed with an eating disorder and 506 of their closest-aged sisters who have never been diagnosed with an eating disorder. The general population sample includes the 506 women diagnosed with an eating disorder (used in the sibling sample) and 1,217 individuals who are not biological related, have not been diagnosed with an eating disorder, but match on five socio-demographic variables (race, ethnicity, age, gender, and religious affiliation).

Study Objective 3: Testing Social Mechanisms to Explain the Link between Eating Disorders and Early Fertility

For this aim, I will utilize Add Health data only. This sample (N=5,037) will compare the parity and of women with eating disorders or disordered eating behaviors (n=1,223) to those who have never been diagnosed with an eating disorder nor engage in disordered eating behaviors (n=3,814).

Significance of Research

This research makes major methodological contributions to the literature. As noted, our understanding of how eating disorders influence fertility is largely based on studies utilizing clinical samples. The use of clinical samples drastically reduces the generalizability of research findings (Cohen and Cohen 1987). To be diagnosed with an eating disorder, an individual must have access, and the desire, to seek the help of a medical professional; this reduces the generalizability of studies that utilize only clinical measures of diagnosis. For example, the literature broadly indicates that racial and ethnic minorities are equally likely to present behavioral symptoms of eating disorders, that is, engage in eating disorder behavior, but may be less likely to be diagnosed (Cachelin et al. 2000; Gordon, Perez, and Joiner 2002). Thus, racial and ethnic minorities are often excluded from eating disorder research because there are so few cases available in the clinical setting.

Conversely, available community studies of eating disorders have faced issues with small proportions of individuals who are diagnosed within their sample (see Tabler and Utz (2015) as an example). In these cases, the sample sizes of those who have been diagnosed with a condition are too small for meaningful within or cross group comparisons. Therefore, asking respondents about disordered eating behaviors that are at risk, or indicative, of eating disorders in community-based surveys is an alternative way of assessing eating disorders that moves beyond utilization of medical services, and may be able to capture individuals who are underrepresented in samples of individuals who are diagnosed with an eating disorder, such as men or racial or ethnic minorities (Woodside et al. 2001). A problem with using disordered eating behaviors as proxy

measures of eating disorders, even though they have been found to have substantial impacts on the lives of individuals who engage in them (Stephen et al. 2014; Tabler and Utz 2015), is that the behaviors may not be as severe or long-term as eating disorders that meet full diagnostic criteria.

Community- or population-based studies with large samples that measure both disordered eating behaviors and eating disorder diagnosis should therefore be pursued, and more importantly, their results should be compared. This study utilizes two data sources: data collected at the community level that includes measures of disordered eating behaviors in addition to self-reported diagnosis, and the population level (of the State of Utah) that includes clinical measures of eating disorders (diagnosis only). Comparing the results generated from analyses of these two diverse samples will improve our understanding of eating disorders, and provides unique insight into how sampling and measurement may influence the inferences we make about eating disorders.

This research also adds important contributions to ongoing eating disorder and fertility research. As stated previously, eating disorder outcome research has focused primarily on physical and mental health outcomes. Fertility outcomes in particular have largely been examined from a biomedical perspective, where fertility is approached as a biological outcome rooted in physical capability. For example, fertility studies have looked at Body Mass Index (BMI), body fat percentage, and irregular menstrual cycles, to assess how eating disorders or disordered eating behavior may reduce or disrupt fertility in women (Freizinger et al. 2010; Jokela, Elovainio, and Kivimäki 2008; Zaadstra et al. 1993). While these studies are important and provide insight into the adverse physical health outcomes of eating disorders, fertility is also a social behavior

that represents an important rite of passage into adulthood: the transition to parenthood (Buchmann 1989). Although we know that many women seeking fertility treatment have experienced or engaged in disordered eating behaviors (Freizinger et al. 2010), we know very little about the overall fertility experiences of women with eating disorders, including their parity and fertility timing.

Increasing our knowledge of the overall fertility experiences of women with eating disorders has several important practice implications. As noted previously, there is evidence to conclude that eating disorders have a genetic and familial basis (Trace et al. 2013). Mothers and fathers with eating disorders may be passing genes and/or perpetuating a social environment that may lead to a transference of disordered eating behaviors to their children (Strober et al. 2000). Understanding the fertility experiences and trends of women with eating disorders is arguably important for practitioners, because childbearing by those with eating disorders has an important influence on the prevalence of eating disorders.

In addition, both infertility and unplanned pregnancy are associated with increased risk of depression, anxiety, and psychosocial conditions (Cousineau and Domar 2007; Gipson, Koenig, and Hindin 2008). Studying the childbearing experiences of individuals with eating disorders and disordered eating behaviors is important because negative fertility experiences could exacerbate concurrent physical and mental health conditions. Conversely, studying the fertility experiences of individuals with eating disorders is important because childbearing may also have a protective effect on their long-term health; Papadopoulos et al. (2013) found that childbearing was associated with 65 percent lower mortality for women with anorexia nervosa. Successful childbearing

may have the ability to mediate some of the negative physical and mental health consequences associated with eating disorders, while negative fertility experiences may exacerbate ongoing health and social issues.

Conclusion

An estimated 20 million American women alive today have or will suffer from an eating disorder (Wade, Keski-Rahkonen, and Hudson 2011), although many women with eating disorders are unlikely to receive treatment (Hudson et al. 2007). Understanding the family formation experiences of individuals with eating disorders is important because it can show that eating disorders affect other types of life opportunities and outcomes associated with adult development, which may further exacerbate the health and mortality outcomes commonly associated with eating disorders. If researchers and clinicians have a better understanding of the life trajectories and potential mechanisms through which individuals with eating disorders may be accruing risk for poor health, in this case, their marriage and fertility behaviors, intervention can be better tailored to address the unique life circumstances of individuals with eating disorders. Future studies should consider eating disorders from a life course perspective, and utilize large, population samples to improve the generalizability of findings. It is important to understand how eating disorders may be acting as a turning point that sets individuals on a different life trajectory whereby they do not have the same opportunities for success throughout adulthood.

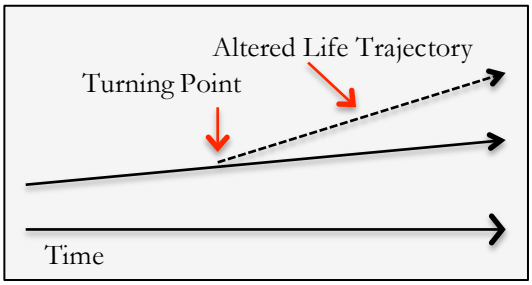

Figure 1.1 Turning Point

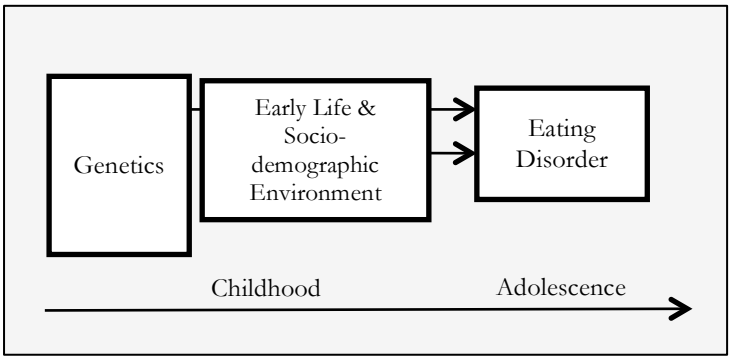

Figure 1.2 Etiology of Eating Disorders

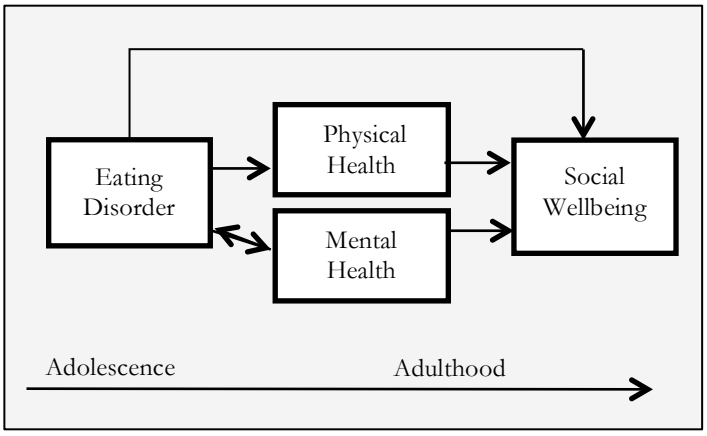

Figure 1.3 Consequences of Eating Disorders

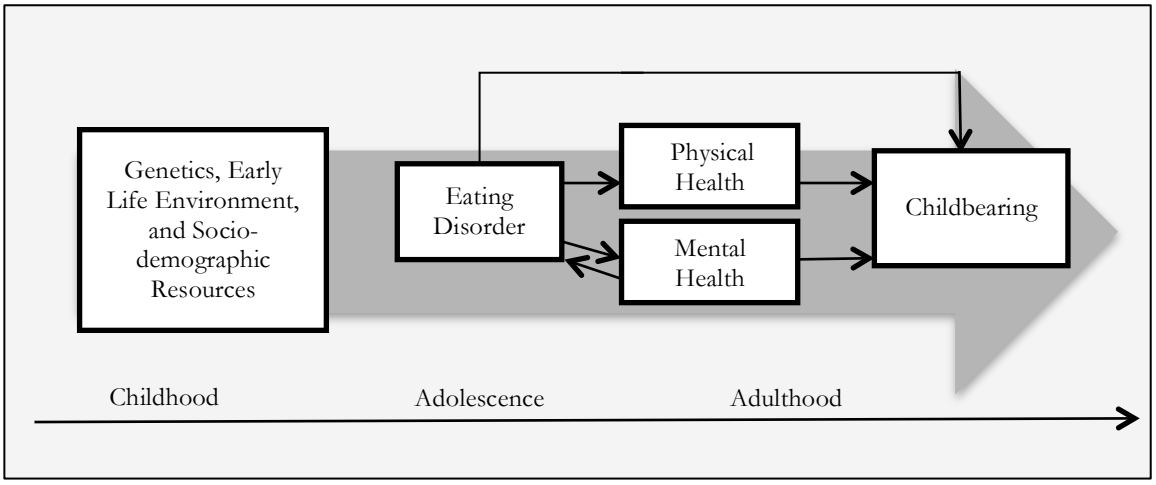

Figure 1.4 Eating Disorders and the Life Course

CHAPTER 2

EATING DISORDERS AND FERTILITY TRAJECTORIES OF TWO SAMPLES OF WOMEN

Introduction

Sampling methodologies, including the size and type of sample, have implications for research outcomes and for the inferences we make about these outcomes (Altman 1990; Dechartres et al. 2013). Small samples may prevent research findings from being extrapolated, whereas large samples may amplify the detection of differences, resulting in an emphasis on statistical differences that may not be clinically or socially relevant (Altman 1990). In addition, sample type is likely to bias the outcome of a research investigation. This is particularly true for clinical sampling, which samples a population *currently* suffering from a disease (a prevalence sample), compared to community or population samples, which tend to represent a population that has *ever* experienced the disease (an incidence sample) (Cohen and Cohen 1984). As Cohen and Cohen (1984) note, clinical samples are therefore biased toward cases of long duration or greater severity, and/or capture individuals actively seeking treatment for an illness or condition, thereby limiting the generalizability of clinical findings to the broader population.

These methodological principles are critically important to understanding the consequences of eating disorders, given that prior research has relied heavily on clinical samples (Kouba et al. 2005; Stewart et al. 1990). While past clinical research has

examined the influence of eating disorders on the biological aspects of fertility, such as fecundability (probability of becoming pregnant in a menstrual cycle) and infertility (inability of a sexually active woman engaging in regular unprotected sexual intercourse to achieve pregnancy in a twelve-month period) (Bulik et al. 1999; Stewart et al. 1990), it is important to study the overall fertility experiences of women with eating disorders beyond the clinical setting. The fertility experiences of women with eating disorders, including their fertility timing (age at first birth) and parity (number of children), have important implications for additional life opportunities across their lifespan, including employment and education (Kessler et al. 1995; Rindfuss et al. 1984), their future health and wellbeing (Papadopoulos et al. 2013), and the health risks posed to their children (Patel et al. 2002; Waugh and Bulik 1999). Therefore, being aware of how sampling and measurement may be shaping the inferences we may be making about the influence of eating disorders is important.

Most prior research examining the influence of eating disorders on fertility has relied on clinical sampling. However, those studies that do rely on population or community samples (Papadopoulos et al. 2013) utilize a clinical *measurement* (i.e., diagnosis) of an eating disorder. To be diagnosed with an eating disorder, an individual must have access, and the desire, to seek the help of a medical professional. For example, racial and ethnic minorities are equally likely to present behavioral symptoms of eating disorders, that is, engage in eating disorder behavior, yet may be less likely to be diagnosed or seek treatment (Cachelin et al. 2000; Gordon et al. 2002; Ham, Iorio, and Sovinsky 2015). Thus, representative samples of racial and ethnic minorities may be excluded from eating disorder research because there are so few cases available. This

reduces the generalizability of the findings of studies that utilize population or community samples, yet still rely on clinical measurement of eating disorders.

In some cases, it is difficult to examine the influence of clinically measured eating disorders within community or population samples (which better represent the general population) given that there are very small subsamples or cases of individuals who are diagnosed with eating disorders within the sample. In these types of studies, the sample sizes of individuals with the condition of interest may be too small for meaningful within or cross group comparisons. This has led scholars to use proxy measures, such as survey scales measuring risk factors for particular conditions or behaviors (Stephen et al. 2014; Tabler and Utz 2015). For example, asking respondents about disordered eating behaviors that are indicative of eating disorders in community-based surveys is an alternative way of assessing eating disorders that moves beyond utilization of medical services, and may be able to capture individuals who are underrepresented in samples of individuals who are diagnosed with an eating disorder, such as men or racial or ethnic minorities (Woodside et al. 2001).

A problem with using disordered eating behaviors as proxy measures of eating disorders, even though they have been found to have substantial effects on the lives of individuals who engage in them (Stephen et al. 2014; Tabler and Utz 2015), is that the behaviors may not be as severe or long-term as eating disorders that meet full diagnostic criteria. Therefore, both clinical and nonclinical sampling designs, and both clinical and nonclinical measurement of eating disorders, have implications for the types of inferences we make about the influence of eating disorders on fertility outcomes.

This study seeks to remedy some of the bias inherent in current eating disorder

and fertility research by comparing two different nonclinical samples of women. This study utilizes two data sources: 1) a nationally-representative sample of women that includes measures of disordered eating behaviors in addition to self-reported diagnosis from the National Longitudinal Study of Adolescent and Young Adult Health (Add Health), and 2) a population-based sample of the State of Utah that includes clinical measures of eating disorders (diagnosis) from the Utah Population Database (UPDB). We examine the age at first birth and parity (number of children) of women within these two samples to assess the influence of eating disorders or disordered eating behavior on fertility trajectories. Comparing the results generated from analyses of these two diverse samples will improve our understanding of how eating disorders influence fertility, and allow greater discussion regarding how sampling and/or measurement may be influencing the inferences we make about eating disorders.

Eating Disorders and Fertility

There is a well-documented link between eating disorders, infertility, and pregnancy difficulties (Bulik et al. 1999; James 2001; Linna et al. 2013; Stewart et al. 1990). Many women seeking fertility treatment have a history of disordered eating behaviors (Freizinger et al. 2010; Stewart et al. 1990). Fertility studies have looked at body fat percentage and menstrual cycle disruption to assess how eating disorders reduce fertility in women (Cousins et al. 2015; Freizinger et al. 2010; Stewart et al. 1990; Stewart 1992). Fertility has therefore been approached as a biological outcome rooted in physical capability within the context of eating disorder research.

While these studies are important and provide insight into the adverse physical health outcomes of eating disorders, fertility is also a social behavior rooted in individual

and societal family preferences (Barber 2000; Michael and Tuma 1985), with parenthood representing an important rite of passage into adulthood (Arnett 2000). Recent community studies, for example, indicate that women with eating disorders are more likely to identify a pregnancy as being unplanned (Bulik et al. 2010; Easter et al. 2011). There are sociological explanations for why women with eating disorders may be at increased risk of unplanned pregnancy; eating disorders and disordered eating behaviors in adolescence are associated with higher levels of delinquency and substance use (Piran and Robinson 2011; Stephen et al. 2014; Striegel-Moore et al. 2003). Such risky behaviors are also commonly associated with risky sexual behavior, including early age of first sexual encounter and higher numbers of sexual partners, and early or unintended pregnancy (Naimi et al. 2003; Pugh et al. 1990; Yamaguchi and Kandel 1987).

Although it is clear based on clinical research that reduced fecundability may occur at a discrete time point as a result of the adverse physical health outcomes of eating disorders, more research is arguably necessary, as very little is known about the overall fertility intentions, experiences, and histories of women with eating disorders across their lifespan, including their parity and fertility timing. Understanding the fertility experiences (i.e., parity and age at first birth) of individuals with eating disorders is important because it can show that eating disorders affect other types of life opportunities associated with adult development, such as employment and education, which may further exacerbate the health and mortality outcomes commonly associated with eating disorders. If researchers and clinicians have a better understanding of the potential mechanisms through which women with eating disorders may be accruing risk for poor health, future intervention can be better tailored to address the unique life circumstances, including their fertility

experiences, of women with eating disorders.

This study examines the fertility timing (age at first birth) and parity (number of children) of women within two separate samples: clinical data available through the Utah Population Database (UPDB), and community data available through the National Longitudinal Study of Adolescent and Young Adult Health. The measurement of eating disorders in the UPDB sample includes eating disorder diagnosis available through medical records, while the measurement of eating disorders within the Add Health sample includes self-reported diagnosis or disordered eating behaviors. I anticipate that the measurement and sampling differences between the UPDB and Add Health data will influence the association between eating disorders and fertility experiences:

First, given that the UPDB sample includes a clinical measurement of eating disorder, I anticipate that the association of eating disorders with fertility will be similar to the results of past clinical studies; women with eating disorders often go undiagnosed with an eating disorder for several months or years, and often times, only after the eating disorder has become severe or life threatening (Yager and Powers 2008). Therefore, the physical health complications commonly associated with eating disorders may be present within this sample of women with eating disorders, thereby leading to reduced (lower overall parity) or delayed (later age at first birth) fertility. Thus, I anticipate that women with an eating disorder diagnosis within the UPDB sample will be more likely to experience a later age at first birth, and lower overall parity by the age of 32, compared to women without an eating disorder diagnosis.

Second, I anticipate that there will be either no association of eating disorders with fertility, or a positive association (i.e., more children, earlier age at first birth),

among women within the Add Health community based sample. Given the use of a proxy measure (disordered eating behavior), the eating disorder experiences of women within the Add Health sample may be less severe or prolonged as the eating disorder behavior of women within the UPDB sample. Thus, underlying social mechanisms associated with both increased fertility and eating disorders, such as deviant behavior and sexual risk-taking, may be present within this subsample of women with eating disorders or disordered eating behaviors, whereas the adverse health outcomes (associated with reduced fertility) may not yet be present. Therefore, I anticipate that women with a self-reported eating disorder diagnosis or behavior will be more likely to experience earlier age at first birth, and higher overall parity by Wave IV (age 24-32), than women without a self-reported eating disorder or disordered eating behavior.

Methods

Data Sources

Add Health

The present study extracts data from the restricted-use (full sample) National Longitudinal Study of Adolescent and Young Adult Health (Add Health) survey Waves I (1994–1995), II (1996), III (2001–2002), and IV (2008-2009). Add Health has been following American adolescents over the course of 14 years, with four waves of data collection since the study's inception when participants were in grades 7–12. Wave I was conducted between 1994 and 1995, with sampling from 132 schools in the United States, representative for region, urbanicity, size, and ethnicity. Initially, 80 high schools were recruited, and each participating school identified a feeder middle school in the community, with the requirement of the inclusion of 7^{th} graders. A total of 20,074

adolescents in grades 7–12 at Wave I were surveyed using self-administered, in-school questionnaires. In-home interviews were conducted with participants at Wave III and Wave IV. Due to attrition or loss to follow-up, Wave IV included approximately 15,700 of the original 20,000 individuals surveyed. Further details about the study design can be found elsewhere (Urdy, Bearman, and Harris 2014).

The Utah Population Database

The UPDB has comprehensive coverage of the Utah population, and contains high-quality socio-demographic, family, medical, and vital records linked into large multigenerational pedigrees. The UPDB contains information on over 8 million individuals. The UPDB is based on linked vital records into multigenerational pedigrees, medical records from the University of Utah Hospitals and Clinics (UUHC) (one of the largest hospital and clinic networks in the State of Utah), statewide hospitalization records provided by the Utah Department of Health (UDOH), and height, weight, and residence information provided by the Department of Motor Vehicles (DMV). Medical, hospitalization, and DMV records are available from 1995-present. This study has been approved by the University of Utah's Resource for Genetic and Epidemiologic Research and its Institutional Review Board.

The UPDB data used for these analyses include women who have ever been treated for an eating disorder. There are 5,760 women captured in the UPDB who have been treated for an eating disorder between 1995 and 2015. These individuals were matched to three randomly selected individuals in UPDB who have never been treated for an eating disorder, who are not biologically related, but match on five key socio-demographic characteristics (i.e., age, sex, race, ethnicity, and religious affiliation) to

generate a comparison population of 17,210 women. This results in a total sample of 22,970 women.

Samples

Add Health

Of those surveyed at Waves I, III, and IV, 8,352 respondents were female. Of these 8,352 women, 2,400 had missing values on key variables, such as race/ethnicity (*n*=642) or BMI (*n*=1,387) or eating disorder or disordered eating behavior (*n*=1296). After list-wise deletion, a sample of 5,952 women was generated. The analytic sample from Add Health includes 1,430 women who self-report having been diagnosed with an eating disorder or engage in eating disorder behaviors at Wave III, and a group of 4,522 female peers without an eating disorder or related behavior. This results in a total analytic sample of 5,952 individuals. In our second set of analyses examining age at first birth, women who have had a child before Wave III are excluded to address potential violations of time-order causality. This yields a sub-analytic sample of 4,061 women, 911 of who have been diagnosed with an eating disorder or engage in eating disorder behaviors, and 3,150 of their unafflicted peers.

Utah Population Database

Of the original 22,970 women captured in the UPDB data set, 1,648 were younger than 15 at time of sampling. An additional 9,960 individuals did not have an available BMI measurement, or BMI was measured after age 32. An additional 2,477 individuals had missing measurement of their median household income (census block level). Of women diagnosed with an eating disorder, some (*n*=2,415) were diagnosed outside the

study time frame (i.e., before age 12 or after age 32 years). After list-wise deletion on relevant covariates, an analytic sample of 12,121 women, approximately 45 percent of the original UPDB sample, was generated. The analytic sample includes 1,725 women who were diagnosed with an eating disorder between 1995 and 2015, and include women diagnosed between the ages of 12-32. This leaves a comparison group of 10,396 individuals without an eating disorder diagnosis. In the second set of analyses examining age at first birth, women who had a child or right-censored before age 18, who were diagnosed with an eating disorder after the birth of their first child, or whose BMI was measured after their first birth, are excluded to have an appropriate comparison to the Add Health sample. This yields a sub-analytic sample of 8,364 women, 1,231 who have been diagnosed with an eating disorder, and a comparison group of 7,133 women who have not been diagnosed with an eating disorder.

Measures

In the following section, the measures from both the Add Health and UPDB samples are described. For ease of comparison, Table 2.1 presents the similarities and differences between measurements across the two samples.

Add Health

Dependent Variables

Parity was measured at Wave IV, when participants were in early adulthood, with the average age of 28 for respondents (Range: 24-33), and is a count of total live births.

Age at first birth was measured in years. For the Cox proportional hazard models, I exclude women whose first birth occurred before Wave III to establish time-order

causality. Right censoring is set to age of subject at Wave IV for nulliparous (childless) subjects.

Primary Independent Variable

Self-identified eating disorder diagnosis was assessed with a single yes/no question, “have you *ever* been diagnosed with an eating disorder?” To select additional individuals who report engaging in disordered eating behaviors, I first identified individuals participating in unhealthy compensatory behaviors directed at maintaining or losing weight. Respondents were asked, “During the past seven days what did you do to keep from gaining weight?” Individuals who reported behaviors, "made your-self vomit, fasted or skipped meals, took laxatives, took diet pills, or diuretics,” were coded as having disordered eating behaviors. I also identified individuals with binge eating symptoms. I included those who identified having “eaten so much in a short period of time that [they] would have been embarrassed if others had seen them do it, in the past seven days” as having a disordered eating behavior.

The measure labeled *Eating Disorder or Disordered Eating Behavior* combines individuals who self-identify as having been diagnosed with an eating disorder with those who engage in unhealthy weight-related compensatory behaviors, and/or exhibit binge eating symptoms. The comparative group includes those who do not identify as having been diagnosed with an eating disorder, nor identify engaging in any of the measured disordered eating behaviors. Survey questions related to eating disorders and disordered eating behaviors were only asked in Wave III.

Covariates

Race and Ethnicity is a categorical variable comparing Non-Hispanic whites, Hispanics, and Non-Hispanic other. Although greater detail on race and ethnicity was available (e.g., Non-Hispanic Asian), there were too few cases to yield meaningful analysis.

Body Mass Index was calculated by first using the height and weight data measured by the interviewer at Wave III. Self-Reported height and weight was used if the respondent had missing measured data (0.1 percent of the original sample).

In addition, I controlled for the household income of respondents at Wave III. *Household Income* is an ordinal category of household income ranges: 1 "<$10,000" 2 "$10,000 to $14,999" 3 "$15,000 to $19,999" 4 "$20,000 to $29,999" 5 "$30,000 to $39,999" 6 "$40,000 to $49,999" 7 "$50,000 to $74,999" 8 "+$75,000."

UPDB

Dependent Variables

Parity is measured as a count of total live births by age 32 taken from vital records (birth certificates). Parity cut-off age and right censoring is set to 32 years of age within UPDB so as to match the right censoring of the Add Health data at Wave IV.

Age at first birth was measured in years, with right censoring set at age 32 or age of death/last age known to live in Utah for those who died or moved before age 32, for childless subjects. For Cox proportional hazard models, I excluded women whose first birth was before age 18 to create an appropriate comparison with the Add Health sample.

Primary Independent Variable

Eating Disorder was measured using hospitalization (UDOH) and outpatient (UUHC) medical records. Individuals with records indicating primary diagnosis of ICD-9 code 307.1 or ICD-10 code F50 (anorexia nervosa), ICD-9 Code 307.51 or ICD-10 code F50.2 (bulimia nervosa), ICD-9 307.50 or ICD-10 Code F50.8-F50.9 (eating disorder-not otherwise specified), were identified as having been diagnosed with an eating disorder diagnosis. I restricted the eating disorder sample to those diagnosed between ages 12-32 years (average age at first diagnosis=21.8 years, standard deviation [*SD*]= 5.32 years).

Covariates

Race and Ethnicity is a categorical variable comparing Non-Hispanic whites, Hispanics, and Non-Hispanic Other, taken from vital records.

Body Mass Index was calculated using height and weight information from individual's hospital records (at time of eating disorder diagnosis) or using earliest available height and weight information of driver's license issued after the age of 16 or before age 32 (average age at BMI measurement= 21.16 years, *SD*=4.98).

In addition, I controlled for average household income at the census block level. UPDB staff constructed average household income at the census block level using available driver's license resident information and census data. This information was only available at single time point for each respondent (average age at Median Income measurement=21.74 years, *SD*=5.86) and was transformed into ordinal categories to match the household income measurement of the Add Health sample; *Household Income* is an ordinal category of eight average household income ranges: 1 "<$10,000" 2 "$10,000 to $14,999" 3 "$15,000 to $19,999" 4 "$20,000 to $29,999" 5 "$30,000 to

$39,999" 6 "$40,000 to $49,999" 7 "$50,000 to $74,999" 8 "+$75,000."

Analytic Plan

In a first step, I present descriptive statistics for both the Add Health and UPDB samples. In a second step, I assess the influence of eating disorders on *Parity* for both the Add Health and UPDB analytic samples using negative binomial regression. In a third step, I assess the influence of eating disorders on *Age at first birth* of both the Add Health and UPDB analytic subsamples (limited to births after Wave III for Add Health, and after 18 for UPDB). I do so using Cox proportional hazard modeling. Cox proportional hazard modeling is a statistical method of analyzing the length of time between "exposure" (eating disorder diagnosis or disordered eating behavior) and "event" (first birth) as the hazard of event occurrence (Cox 1992). It has the advantage of analyzing events that occurred (i.e., first birth), without discarding information on those who have yet to experience an event. I stratify the Cox proportional hazard models by birth year (i.e., each birth year being allowed to have their own baseline hazard) to control for potential confounding cohort effects.

Results

Table 2.2 reports descriptive statistics of the Add Health analytic sample. Table 2.3 reports descriptive statistics of the UPDB analytic sample. Approximately 22 percent of the Add Health analytic sample reports an eating disorder or disordered eating behavior at Wave III. Fifteen percent of the UPDB analytic sample had been diagnosed with an eating disorder after 12, but before age 32.[1] Approximately 62 percent of the Add

[1] It is important to note that this does not represent the true proportion of individuals in the State of Utah diagnosed with an eating disorder; this study uses a 3:1 match random sampling design.

Health sample is non-Hispanic white, 9 percent is Hispanic, and 29 percent is non-Hispanic other. Eighty-seven percent of the UPDB sample is non-Hispanic white, 9 percent is Hispanic, and 4 percent is non-Hispanic other. The median annual household income bracket of women in the Add Health sample is $20,000-29,999 (at Wave III). The median average household income bracket (at census block level) of women in the UPDB analytic sample was $40,000-49,000. The average age at measurement of household income for UPDB sample was 22 years (*SD*=5.8). The median number of children born to women in the Add Health sample by early adulthood (Wave IV, age range 26-32) was one, while the median number of children born to women of the UPDB sample by early adulthood (at age 32) was two. The average age at first birth of women in the Add Health analytic subsample (limited to first births after age at Wave III, age range 18-24) at Wave IV is 24.7 years (*SD*=2.78), while the average age at first birth of the UPDB analytic subsample (limited to first births after age 18, but before 32) is 24.5 (*SD*=4.31).

Table 2.4 (column 1) reports the negative binomial regression results for the Add Health analytic sample; Eating disorder or disordered eating behavior has a *positive* effect on parity (count of births) (incidence rate ratio (*IRR*)=1.14 [95 percent Confidence Interval= 1.07-1.21]; $p<0.001$), when controlling for race/ethnicity, marital status by age 32, household income, BMI, and birth year. Table 2.4 (column 2) reports the negative binomial regression results for the UPDB analytic sample; eating disorder diagnosis (between the ages of 12-24) has a *negative* effect on parity (*IRR*=0.40 [0.38-0.43]; $p<0.001$) when controlling for all else.

True lifetime prevalence is estimated between 1-5 percent of the US female population: Hudson, James I., Eva Hiripi, Harrison G. Pope, and Ronald C. Kessler. 2007. "The Prevalence and Correlates of Eating Disorders in the National Comorbidity Survey Replication." *Biological Psychiatry* 61(3):348-58.

Table 2.5 (column 1) reports the Cox proportional hazard model results for the Add Health analytic subsample.[2] These results indicate that eating disorder or disordered eating behavior in adolescence and young adulthood is associated with *increased* risk of first birth after Wave III (hazard rate ratio (*HRR*)=1.16 [1.03-1.30]; $p<0.001$) when controlling for race/ethnicity, marital status, household income, BMI, and birth year. Table 2.5 (column 2) reports the Cox proportional hazard model results for the UPDB analytic subsample; eating disorder diagnosis is associated with *decreased* risk of first pregnancy after the age of 18 (*HRR*=0.35 [0.31-0.39]; $p<0.001$) when controlling for race/ethnicity, marital status, average household income at the census block level, BMI, and birth year.

Discussion

This study has two major findings: first, eating disorders influence the fertility experiences of women, including their parity and age at first birth, and second, eating disorder measurement and sampling design shapes inferences about the influence of eating disorders on fertility.

Although steps were taken to transform the UPDB and Add Health analytic samples (which were drawn using very different sampling designs) into comparable samples, including the use of similar covariates across the data sources and limiting the observation period of the UPDB analytic sample, there are important differences in the

[2] There is selection bias occurring within the Add Health subsample, where women who report eating disorder diagnosis or behavior are less likely to be included in survival analyses (see Table 2.2). This is due to the fact that women were excluded if their eating disorder or behavior was measured *after* the birth of their first child. Despite the exclusion of these women with earlier births, results still indicate that women within the Add Health subsample with self-reported eating disorder diagnosis or disordered eating behavior have higher risk of first birth than unafflicted peers.

measurement of eating disorders within these two samples. The UPDB data represent a clinical sample with a general population comparison (Utah only), while the Add health data represent a nationally representative longitudinal cohort sample. The measurement of eating disorders in the UPDB sample includes eating disorder diagnosis available through medical records, while the measurement of eating disorders within the Add health sample includes self-reported diagnosis or disordered eating behaviors.

The measurement and sampling differences between the UPDB and Add Health data shaped the results of this study. Women with an eating disorder diagnosis within the UPDB sample were more likely to experience later age at first birth, and lower overall parity by the age of 32 years, compared to women without an eating disorder diagnosis, while women with a self-reported eating disorder diagnosis or eating disorder behavior within the Add Health sample were more likely to experience earlier age at first birth, and higher overall parity by Wave IV (age 24-32 years), than women without a self-reported eating disorder or disordered eating behavior.

Differences in results between the two samples illustrate that although the use of disordered eating behaviors or other symptomology may be useful as proxy measures of eating disorders, there are likely to be important differences in the outcomes of studies examining disordered eating behaviors compared to studies examining clinical diagnoses. One explanation of the differences in fertility outcomes between these two forms of measurement may be that the eating disorder behavior of women within the Add Health sample may be less severe or prolonged as compared to the eating disorder behavior of women within the UPDB sample.

Individuals often go undiagnosed with an eating disorder for several months or

years, and often times, only after the eating disorder has become severe or life threatening (Yager and Powers 2008). Approximately 40 percent of individuals within the UPDB sample were first diagnosed with an eating disorder at the time of hospitalization (inpatient), and 30 percent of the UPDB sample diagnosed with an eating disorder sample experienced an additional eating disorder-related medical event (inpatient or outpatient) after their first event. This may indicate that women with an eating disorder within the UPDB sample may experience additional physical and mental health complications commonly associated with eating disorders that may be disrupting their fertility, such as weight-related and nutrition-related menstrual disruption (Langley 2014; Reid and Van Vugt 1987), or reduced ovarian function associated with long-term depression (Harlow et al. 2003).

It is likely that women within the Add Health sample with self-reported eating disorder or disordered eating behavior may not be experiencing the aforementioned physical or mental health mechanisms that may explain the reduced fertility of women diagnosed with an eating disorder within the UPDB sample. Women with self-reported eating disorder or disordered eating behavior within the Add Health sample have higher BMIs (28.78, *SD*=7.67), for example, than women with eating disorders within the UPDB sample (22.18, *SD*=8.57).

In addition, unlike UPDB, Add Health data are more prone to response or participation biases. For example, women with more severe eating disorders may have been excluded from the Add Health sample entirely given research indicating that individuals with psychiatric disorders are more likely to drop out of *survey*-based research studies (de Graaf et al. 2000). Although preliminary analyses (not shown) did

not indicate that women with self-reported eating disorder diagnosis or behavior at Wave III were more or less likely to present at Wave IV, it is unknown whether women experiencing eating disorders at an earlier point in adolescence (e.g., before Wave III) were less likely to have participated in subsequent surveys.

In addition, the samples here are representative of different populations. The UPDB contains comprehensive information regarding the Utah population. The Utah population has the highest total fertility rate in the United States, estimated at 2.3 births per woman (Martin et al. 2015). Given the high fertility rates experienced by women in Utah, it is possible that the effects of eating disorders on fertility may be more pronounced relative to populations that have more reduced fertility trends (e.g., Rhode Island, which has an estimated total fertility rate of 1.5 births per woman). As anticipated given the high fertility of women within Utah, women within the Add Health sample have a lower average number of total births (1.08, *SD*=1.19) compared to the Utah sample (1.73, *SD*=1.73).

Despite the potential for data bias, there are mechanisms that may explain why we see increased and early fertility of women with eating disorder or disordered eating behavior; disordered eating behaviors have been conceptualized as a form of internally directed deviance, resulting from negative self-feelings (Sischo et al. 2006). In addition, eating disorders and disordered eating behaviors have been associated with forms of externalized deviance; for example, eating disorders and disordered eating behaviors in adolescence are also associated with higher levels of delinquency and substance use (Piran and Robinson 2011; Stephen et al. 2014; Striegel-Moore et al. 2003), such risky behaviors are also commonly associated with risky sexual behavior, including early age

of first sexual encounter and higher numbers of sexual partners, and early or unintended pregnancy (Naimi et al. 2003; Pugh et al. 1990; Yamaguchi and Kandel 1987).

Furthermore, research indicates that young adults diagnosed with one or more psychiatric disorders, including depression, eating disorders, anxiety, and antisocial disorder, are more likely to engage in risky sexual intercourse (noncondom use, higher number of partners) and have sexual intercourse at an early age (Ramrakha et al. 2000; Shrier et al. 2001). Young women with eating disorder or disordered eating behavior may be more likely to engage in risky sexual behaviors, including earlier ages of first sexual intercourse and higher number of sexual partners, due to their comprised self-esteem (Fisher et al. 1991).

In addition, women with eating disorders or disordered eating behavior may be less likely to use effective forms of contraception due to their assumption that they may be infertile due to their compromised health status (Bulik et al. 2010; Downs et al. 2004), resulting in higher rates of unplanned pregnancy (Bulik et al. 2010; Easter et al. 2011). Because unintended pregnancy is a risk factor for subsequent, unintended pregnancy (Kuroki et al. 2008), women with eating disorders or disordered eating behavior may not only be at risk of early entry into parenthood, but of experiencing multiple births at a younger age than their unafflicted peers, like the women with eating disorders or disordered eating behavior within the Add Health sample.

There may be two sets of competing mechanisms, physical versus social, that influence the fertility experiences of women with eating disorders. Additional research is necessary to identify the social mechanisms in particular that may be influencing the altered family formation experiences of women with eating disorders or disordered eating

behaviors.

There are several limitations of this study. First, this study does not account for the full fertility history of women (between the ages of 15-50 years of age), due to data constraints within the Add Health sample that allows for an examination of a more limited time frame (roughly between the ages of 18-32 for both the Add Health and UPDB samples). The results of this study speak to the fertility of women in early adulthood, rather than completed fertility. It is important to note, however, that in supplementary analyses (not shown) of the UPDB samples, results do not substantially change when using full fertility age range (15-50 years of age). Given the historical trend toward delayed fertility in the United States, particularly increased fertility after the age of 40 (Mathews and Hamilton 2009; Whelpton, Campbell, and Patterson 2015), future studies should examine complete fertility histories of women with eating disorders.

In addition, this study does not account for the potential mechanisms that may explain the link between eating disorders and fertility, beyond BMI; although steps were taken to transform variables within the UPDB and Add Health analytic samples into acceptable cross-sample comparisons, differences in type and number of measures between the data sources limited the amount of covariates that could be included in the analyses. For example, there is information on the delinquency experiences and sexual orientation of women in the Add Health sample, but not the UPDB sample. Future studies should attempt to account for additional contextual factors and outcomes commonly associated with eating disorders.

Conclusion

It is well known that eating disorders influence the fertility experiences of women with eating disorders. However, this study illustrates that eating disorder measurement and sampling design shapes the inferences we may be making about the influence of eating disorders on fertility. In addition, there may be two sets of competing mechanisms, physical versus social, that influence the fertility experiences of women with eating disorders. Additional research is necessary to identify the social mechanisms in particular that may be influencing the altered family formation experiences of women with eating disorders or disordered eating behaviors.

Table 2.1 Comparison of Add Health and UPDB Measures

	Add Health	UPDB	Parity or Timing Analyses
Independent Variable			
Eating Disorder	Includes women who self-report ever having been diagnosed, or engage in at least one eating disorder behavior between the ages of 18-26 (Wave III). Comparison group includes women without eating disorder or disordered eating behavior.	Includes women who have at least one outpatient or in-patient (hospitalization) record of eating disorder diagnosis of anorexia nervosa, bulimia nervosa, or eating disorder-not otherwise specified, after the age of 12, but before the age of 32. Comparison group is a random sample of women matched on age and race/ethnicity who have not had an eating disorder diagnosis or event. Timing analysis excludes individuals diagnosed with an Eating disorder after the birth of their first child/right censor.	Both
Dependent Variables			
Number of Children	Total number of children born to women within the sample by ages 24-32 (Wave IV).	Total number of children born to women within the sample by age 32.	Parity analysis only
Age at First Birth	Measured in years at first birth by Wave IV (ages 24-32). Excludes women whose first birth occurred before Wave III (ages 18-26). Right censoring is set to age at Wave IV for childless women.	Measured in years at first by age 32. Excludes women whose eating disorder diagnosis occurred before birth of first child. Right censoring is set to age 32 or age last known to be in Utah for childless women.	Timing analysis only
Control Variables			
Race/ Ethnicity	Compares Non-Hispanic Whites to Hispanics, and non-Hispanic other.	Compares Non-Hispanic Whites to Hispanics, and non-Hispanic other.	Both
Married Before First Child	Compares those who were not married before the birth of their first child or age at censor to women who were born before the birth of their first child or age at censor.	Compares those who were not married before the birth of their first child or age at censor to women who were born before the birth of their first child or age at censor.	Timing analysis only
Marital Status	Compares those who were married by Wave IV (ages 24-32) to those who were not married by Wave IV.	Compares those who were married by age 32 to those who were not married by age 32.	Parity analysis only
Body Mass Index	Calculated using measured height and weight information at Wave III (ages 18-26). For those with missing measurement data, self-reported height and weight information provided at Wave III was utilized.	Calculated using clinically measured height and weight information at first eating disorder measurement by age 32. For those missing clinical measurement of height and weight, driver's license data was utilized. Timing analysis excludes individuals whose only known height and weight information was gathered after first birth or right censoring.	Both
Household Income	*Household Income* is an ordinal category of self-reported annual household income ranges: 1 "<$10,000" 2 "$10,000 to $14,999" 3 "$15,000 to $19,999" 4 "$20,000 to $29,999" 5 "$30,000 to $39,999" 6 "$40,000 to $49,999" 7 "$50,000 to $74,999" 8 "+$75,000." Measured at Wave III (ages 18-26).	*Household Income* is an ordinal category of eight average annual household income ranges measured at the census block level: 1 "<$10,000" 2 "$10,000 to $14,999" 3 "$15,000 to $19,999" 4 "$20,000 to $29,999" 5 "$30,000 to $39,999" 6 "$40,000 to $49,999" 7 "$50,000 to $74,999" 8 "+$75,000."	Both

Table 2.2 Descriptive Statistics of Add Health Analytic Sample

	Full analytic sample (*N*=5,951)	Analytic subsample[d] (*N*=4,061)
	% or Mean (*SD*)	% or Mean (*SD*)
Independent Variable		
Diagnosed with eating disorder[a]		
No, never been diagnosed	96.35%	96.40%
Yes, have been diagnosed	3.65%	3.60%
Disordered eating behavior		
No, disordered eating behavior	77.76%	79.41%
Yes, disordered Eating Behavior	22.24%	20.59%
Eating disorder or disordered eating behavior		
No	75.97%	77.57%
Yes	24.03%	22.43%
Dependent Variables		
Number of children[b]	1.08 (1.19)	0.58 (0.89)
Age at first birth	22.16 (3.69)	24.75 (2.82)
Control Variables		
Race/Ethnicity[c]		
Non-Hispanic white	61.96%	65.23%
Hispanic	8.67%	7.73%
Non-Hispanic other	29.37%	27.04%
Married before first child (or censor)		
Yes	38.27%	55.21%
No	61.73%	44.79%
Marital status[b]		
Never been married	46.22%	52.30%
Married at least once	53.78%	47.70%
Body Mass Index (BMI)[a]	26.90 (7.05)	26.45 (7.03)
Household income[a]		
<$10,000	31.54%	33.00%
$10,000-14,999	8.55%	7.98%
$15,000-19,999	7.91%	7.14%
$20,000-29,999	12.80%	11.40%
$30,000-39,999	9.78%	8.62%
$40,000-49,999	7.38%	7.12%
$50,000-74,999	11.19%	11.80%
$75,000+	10.85%	12.95%

Notes: data come from Add Health.
[a]Measured at Wave III, when respondents were between the ages of 18-24 years of age
[b]Measured at Wave IV, when respondents were between the ages of 24-32 years of age
[c]Measured at Wave I, when respondents were between the ages of 11-18 years of age
[d]Excludes women whose first birth occurred before Wave III

Table 2.3 Descriptive Statistics of UPDB Analytic Sample

	Full analytic sample (*N*=12,121)	Analytic subsample[d] (*N*=8,364)
	% or Mean (*SD*)	% or Mean (*SD*)
Independent Variable		
Diagnosed with eating disorder (ED) [a]		
No, never been diagnosed	85.77%	85.28%
Yes, have been diagnosed	14.23%	14.72%
Dependent Variables		
Number of children [b]	1.73 (1.73)	1.41 (1.65)
Age at first birth	22.88 (3.91)	24.27 (3.61)
Control Variables		
Race/ethnicity		
Non-Hispanic white	87.29%	89.81%
Hispanic	8.65%	6.52%
Non-Hispanic other	4.07%	3.67%
Married before first child (or censor)		
Yes	39.88%	39.59%
No	60.12%	60.41%
Marital status [c]		
Never been married	54.92%	59.52%
Married at least once	45.08%	40.48%
Body Mass Index (BMI)	22.84 (5.36)	22.53 (5.46)
Average household income (census block)		
<$10,000	0.02%	0.01%
$10,000-14,999	0.04%	0.04%
$15,000-19,999	1.05%	0.98%
$20,000-29,999	10.67%	10.63%
$30,000-39,999	21.85%	21.23%
$40,000-49,999	21.57%	21.05%
$50,000-74,999	35.43%	35.69%
$75,000+	9.38%	10.37%

Notes: data come from the Utah Population Database (UPDB)
[a] Includes women diagnosed with an eating disorder (ICD-9 codes 307.1, 307.5-307.52; ICD 10 codes f50-f50.9) between the age of 12-32.
[b] Total live births by age 32
[c] Married by age 32
[d] Excludes women diagnosed with an eating disorder or with a BMI measurement after the birth of their first child or age at right censor

Table 2.4 Negative Binomial Regression Results (Incidence Rate Ratios)

Number of Births	Add health analytic sample	UPDB analytic sample
Primary Independent Variable		
Eating disorder[a]	1.14***	0.40***
	(0.04)	(0.01)
Control Variables		
Race/ethnicity[b]		
Hispanic	1.18***	1.74***
	(0.05)	(0.05)
Non-Hispanic other	1.36***	1.15**
	(0.04)	(0.05)
Marital status[c]	2.12***	2.01***
	(0.07)	(0.04)
Body Mass Index (BMI)	1.01***	1.00
	(0.00)	(0.00)
Household income[d]	0.96***	0.94***
	(0.01)	(0.01)
Birth year	0.94***	0.97***
	(0.01)	(0.00)
N	5,951	12,121
Pseudo R-Squared	0.05	0.09

Notes: data come from Add Health & from the Utah Population Database (UPDB). Robust Standard Errors in Parentheses. Incidence Rate Ratios reported.
*p<0.05 **p<0.01 ***p<0.001

[a]Compares those who identify as having an eating disorder or disordered eating behavior to their unafflicted peers (reference group) within the Add Health sample; compares those diagnosed with an eating disorder (ICD9 codes 307.1, 307.5-307.51; ICD10 F50, 50.2, 50.8, 50.9) to women without an eating disorder diagnosis

[b]Reference group is non-Hispanic white

[c]Compares those who have never been married (reference group) those who have been married at least once by Wave IV (Add Health sample) or age 32 (UPDB sample)

[d]Higher scores indicate higher household income level (ordinal category) measured at the individual level within the Add Health sample, and at the census block level within the UPDB sample.

Table 2.5 Cox Proportional Hazard Results (Hazard Rate Ratios)

Age at First Birth	Add Health subsample	UPDB subsample
Primary Independent Variable		
Eating disorder[a]	1.16**	0.35***
	(0.07)	(0.02)
Control Variables		
Race/ethnicity[b]		
Hispanic	0.98	1.93***
	(0.09)	(0.11)
Non-Hispanic other	1.15*	0.84*
	(0.07)	(0.07)
Marital status[c]	2.26***	2.18***
	(0.12)	(0.07)
Body Mass Index (BMI)	1.00	0.98***
	(0.00)	(0.00)
Household income[d]	1.00	0.90***
	(0.00)	(0.01)
N	4,061	8,364

Notes: data come from Add Health. Robust Standard Errors in parentheses; Hazard Ratios reported; Hazard estimates are stratified by Birth Year
*p<0.05 **p<0.01 ***p<0.001

[a]Compares those who identify as having an eating disorder or disordered eating behavior to their unafflicted peers (reference group) within the Add Health sample; compares those diagnosed with an eating disorder (ICD9 codes 307.1, 307.5-307.51; ICD10 F50, 50.2, 50.8, 50.9) to women without an eating disorder diagnosis

[b]Reference group is non-Hispanic white

[c]Compares those who were married before the birth event or right censor to those who were not married before birth event or right censor (reference group)

[d]Higher scores indicate higher household income level (ordinal category) measured at the individual level within the Add Health sample, and at the census block level within the UPDB sample.

CHAPTER 3

FAMILIAL ENVIRONMENT AND THE INFLUENCE OF EATING DISORDER TYPE ON FERTILITY TRAJECTORIES

Eating Disorders and Fertility

Eating disorders represent a serious and often fatal chronic mental illness that disproportionately affects the health and wellbeing of women (Polivy and Herman 2002). Eating disorders have one of the highest estimated mortality rates (4-6 percent) of any mental disorder (Crow et al. 2009; Sullivan 1995), are predictive of suicide, suicide attempts, and self-inflicted injury (Chesney et al. 2014; Preti et al. 2011; Rosling et al. 2011), and are associated with additional comorbid mental health conditions, including depression and substance abuse (Krahn 1991; Piran and Robinson 2011; Santos et al. 2007).

There is a well-documented link between eating disorders, infertility, and pregnancy difficulties (Bulik et al. 1999; James 2001; Linna et al. 2013; Stewart et al. 1990). Many women seeking fertility treatment have a history of disordered eating behaviors (Freizinger et al. 2010; Stewart et al. 1990). Fertility studies have looked at body fat percentage and menstrual cycle disruption to assess how eating disorders reduce fertility in women (Cousins et al. 2015; Freizinger et al. 2010; Stewart et al. 1990; Stewart 1992).

While fertility research indicates that the physical and mental health

complications associated with eating disorders influence fertility, most fertility research in the eating disorders literature has focused on *anorexia nervosa* (characterized by an obsessive desire to lose weight, primarily by restricting food intake), in which low body weight, suicidal behavior, and mortality rates are particularly high (Yager and Andersen 2005). Women with anorexia are more likely to experience menstrual disruption as a complication of low body weight (Katz and Vollenhoven 2000), miscarriages (Linna et al. 2013), and infertility (inability to become pregnant within 12 months of non-contraceptive use) (Stewart et al. 1990), than women without eating disorders. Given these adverse fertility outcomes, it is surprising that Bulik et al. (1999) found that women with anorexia did not experience difference in their age at first birth, compared to women without eating disorders.

There is also evidence to suggest that women with *bulimia nervosa* (characterized by an obsessive desire to lose weight, coupled with periods of binging followed by extreme exercise, self-induced vomiting, purging, laxative, or diuretic use) and *eating disorder-not other wise specified* (a residual DSM-IV diagnosis for patients with significant features of eating disorders not meeting criteria for anorexia nervosa or bulimia nervosa, referred hereafter as eating disorder-NOS[3]) experience higher rates of miscarriage and are more likely to be childless than women without eating disorders (Linna et al. 2013).

The literature on fertility in eating disorders has several limitations, most pertinent to this study being the relatively small numbers of studies that compare fertility

[3] Although the eating disorder-NOS category has seen changes with the introduction of the DSM-5 in 2013, most notably the official recognition of binge eating disorder, this study uses ICD-9 codes (from diagnoses between 1995-2015) to identify eating disorder types. ICD-9 does not differentiate binge eating disorder within the eating disorder-NOS category (ICD-9 code 351.50).

experiences by eating disorder type (such as Linna et al. (2013)). In addition, most studies, but not all, use short follow-up periods, rather than examining the full fertility trajectories of women with eating disorders (typically understood as ages 15-50). Although clinical research suggests that reduced fecundability (probability of becoming pregnant in a menstrual cycle) may occur as a result of the adverse physical health outcomes of eating disorders, more research is arguably necessary, as very little is known about the long-term consequences of eating disorders across the lifespan, and in particular, whether fertility experiences—parity (total number of children) and fertility timing (e.g., age at first birth)—vary be eating disorder type.

Sibling Study Design

Eating disorders are known to be the result of genetic and epigenetic factors (Campbell et al. 2011). Twin and sibling studies have focused primarily on disentangling genetic and known social etiological risk factors of eating disorders by examining "discordant" siblings. In a sibling discordance study, outcomes or experiences among siblings who are "discordant" for an exposure of interest (e.g., one has an eating disorder, and the other does not) are compared (Donovan and Susser 2011). In these studies, siblings are assumed to be share basic aspects of family context (for example, family socioeconomic status, religious environment, single-parent household, etc.) as well as half their genome (Donovan and Susser 2011).

For example, in a previous study of 45 discordant sister-pairs (one sister with anorexia, one without) authors compared the sisters experiences and found that the sibling with anorexia had different levels of personal vulnerability and exposure to high parental expectations (Karwautz et al. 2001). The authors also found that there were

similarities between the sisters, namely on genotypes and alleles of receptor genes that may contribute to eating disorders, and risk of dieting (Karwautz et al. 2001). A review of eating twin studies highlights the importance of nonshared environmental influences to explain important differences in the development of an eating disorder in one sibling versus another, including differential experiences with body weight teasing, peer group experiences, and life events (Klump et al. 2002).

Sibling studies illustrate that there are shared and unshared characteristics and experiences of discordant siblings, which may explain why one sibling may be more at risk for developing an eating disorder than another, and highlight the methodological value of a sibling study designs. Sibling analyses are particularly useful in testing the robustness of causal relationships that have been identified and confirmed in studies of unrelated individuals (Donovan and Susser 2011). For example, in studies utilizing unrelated samples, it is clear that eating disorders likely reduce fertility. However, these studies do not account for potential confounding genetic or familial environmental factors. Although sisters tend to have more similar fertility experiences than compared to the general population, as a result of shared family factors (Axinn, Clarkberg, and Thornton 1994; Lyngstad and Prskawetz 2010), no eating disorder study to my knowledge has used a sibling design to examine differences in fertility experiences between sisters.

This study uses a sibling comparison design, in particular, a matched discordant sibling pair design, which pairs an eating disorder patient with her closest aged sister not diagnosed with an eating disorder. Biological siblings share similar genetic traits and generally develop in similar early life environments. Comparing individuals who share

some underlying genetic similarity and familial environment will allow us to isolate more definitively the influence of eating disorders on family formation from the potential confounding effects of the early life familial environment. This study also includes a general population comparison sample as a case-control design and robustness check that allows for evaluation of the similarity or differences experienced by siblings. Based on literature indicating that eating disorders reduce female fertility, I hypothesize that while sisters may have more similar fertility experiences, fertility of women with any type of eating disorder (anorexia, bulimia, or eating disorder-NOS) will be significantly lower and delayed compared to their discordant sisters despite their shared early life environment and genetic factors.

In addition, this study directly compares the fertility experiences of women with anorexia, bulimia, and eating disorder-NOS within an analytic sample of women with eating disorders, in order to accurately assess differences in fertility experiences by eating disorder type. Based on literature indicating that the physical health consequences of anorexia are potentially more severe than those experienced by women with bulimia or eating disorder-NOS (Arcelus et al. 2011; Herzog et al. 1999; Mitchell and Crow 2006), I hypothesize that women with anorexia will experience more severe reductions in their fertility compared to women with bulimia or eating disorder-NOS.

Methods

Data

A sibling comparison design is possible through the use of the Utah Population Database (UPDB), a unique data source that links high-quality socio-demographic, medical, and vital records to large multigenerational pedigrees. The UPDB is

comprehensive of the Utah population, and contains information on over 8 million individuals. UPDB staff link vital records to multigenerational pedigrees (family trees), medical records provided by the University of Utah Hospitals and Clinics (UUHC) (one of the largest hospital and clinic networks in the State of Utah), statewide hospitalization records provided by the Utah Department of Health (UDOH), and height, weight, and residence information provided by the Department of Motor Vehicles (DMV). Medical, hospitalization, and DMV records are available from 1995-present. The University of Utah's Institutional Review Board and Resource for Genetic and Epidemiologic Research, which governs data use through the UPDB, has approved this study.

Samples

Eating Disorder Sample

The UPDB data used for these analyses include females who have ever been treated for an eating disorder (anorexia nervosa, bulimia nervosa, or eating disorder-NOS). Individuals captured in the UPDB have been treated for an eating disorder between 1995 and 2015. The full sample of women with an eating disorder included 3,995 women diagnosed with an eating disorder before the birth of their first child and between the ages of 12-50 years. Of this original sample, 1,758 women did not have a BMI measurement before the birth of their first child, or age last known in Utah/age 50 for childless women. Another 675 women had missing values on other key covariates (e.g., race/ethnicity, median household income), resulting in an analytic sample of 1,565 women diagnosed with an eating disorder. Women sampled were born between 1952-2000 (average age at end of study=30.84 years, *SD*=9.24).

Sibling Sample

The original women sampled diagnosed with an eating disorder were matched to their closest-aged sister not diagnosed with an eating disorder. Of the sample of women with an eating disorder without missing data (*n*=1,565), only 1,014 had a sibling match. Of those 1,014 sisters, 511 did not have an appropriate BMI measurement, had missing fertility information, or missing values on other key covariates. This left a total sample of 503 discordant sister pairs (or total sibling sample of 1,006 women).

General Population Sample

The original sample of women diagnosed with an eating disorder were matched to three randomly selected individuals from the Utah population who have never been treated for an eating disorder, who are not biologically related, but match on five key socio-demographic characteristics (i.e., birth year, sex, race, ethnicity, religious affiliation). In order to avoid bias of fluctuating sample sizes, this study compares the same group of women with eating disorders in the sibling-sample (*n*=503) to their randomly generated general population matches (*n*=1,509). Of the general population sample, 292 had missing values on key variables, BMI in particular. This results in a final general population analytic subsample of 1,217 women without an eating disorder, and 503 women with an eating disorder.[4]

[4] In supplementary analyses (not shown) using the full analytic sample of women diagnosed with an eating disorder (*n*=1,565) and a full general population analytic sample, results do not differ significantly compared to the use of this smaller, limited sample.

Measures

Dependent Variables

Parity is measured as a count of total live births between the ages of 15-50 taken from vital records (birth certificates).

Age at first birth was measured in years, starting at age 15, with right censoring set at age 50 or age of death/last age known to live in Utah for those who died or moved before age 50, for childless subjects.

Primary Independent Variable

Eating Disorder is a four-category measure using hospitalization (UDOH) and outpatient (UUHC) medical records. The measure compares those without an eating disorder diagnosis "0" to individuals with records indicating primary diagnosis of anorexia nervosa (ICD-9 code 307.1) "1", bulimia nervosa (ICD-9 Code 307.51) "2", and eating disorder-NOS (ICD-9 307.50) "3". This measure is restricted to those within the eating disorder categories to those diagnosed at age 12 or older, but before the birth of their first child/age at censor (average age at first diagnosis= 23.33 years; standard deviation [*SD*]= 7.98 years).

Covariates

Race and Ethnicity is a categorical variable comparing non-Hispanic whites, Hispanics, and non-Hispanic other, taken from vital records.[5]

Body Mass Index (BMI) was calculated using height and weight information from individual's hospital records (at time of eating disorder diagnosis) or using height and

[5] Although there is additional detail on race and ethnicity within the UPDB data, the counts of these categories (e.g., non-Hispanic black) are too small for meaningful comparison.

weight information or driver license issued after the age of 16 or before age 50 years. Individuals whose BMI was measured after the birth of their first child, or age 50 years, were excluded from these analyses.

In addition, average household income is controlled for (measured before the birth of first child/age 50 years/last age known in Utah) at the census block level in analyses using the eating disorder analytic sample only.[6] UPDB staff constructed a continuous measure of average household income at the census block level using available driver's license resident information and census data.

Suicide attempt/self-inflicted injury event compares those who were treated (between 1995-2015) for suicide attempt/self-inflicted injury (ICD-9 codes E950-958.9) based on medical records before the birth of first child/age at right censor, to those who were not treated. The measure of self-injurious behavior and suicide attempt seeks to control for the most severe behavioral health issue that is highly correlated with eating disorders that may help explain the relationship between eating disorders and reduced fertility (Paul et al. 2002; Preti et al. 2011). Suicidality is most severe among women with anorexia nervosa in particular, and it is therefore important to address whether self-injurious behavior/suicidality potentially mediates the relationship between eating disorder types and fertility (Marzuk et al. 1997; Pompili et al. 2004; Preti et al. 2011).

Analytic Plan

In a first step, I present the descriptive statistics of the sibling pairs, and the full eating disorder analytic samples. In a second step, I compare the age at first birth of

[6] Median household income is excluded from sibling and general population comparisons due to a high volume of missing values. In supplementary analyses (not shown) including median household income, the main effect of eating disorder on parity outcomes did not change substantively.

women with eating disorders to that of a matched sister (closest aged) without an eating disorder using Cox proportional hazard models. Cox proportional hazard modeling is a statistical method of analyzing the length of time between "exposure" and "event" (in this case, first birth) as the hazard of event occurrence (Cox 1992). It has the advantage of analyzing events that occurred (i.e., first birth), without discarding information on those who have yet to experience an event. Cox proportional hazard models are stratified by sibling-pair (i.e., each sibling-pair has their own calculated baseline hazard) to better account for the shared experiences of siblings.

In a third step, I compare the parity of women with eating disorders to that of their matched sister without an eating disorder using negative binomial regression. These models are adjusted for clustering by sibling pair. Negative binomial regression was selected to assess the influence of eating disorder on our count variable, total number of children, over other Generalized Linear Models based on a series of Poisson goodness of fit tests ($p<0.001$), and likelihood ratio tests, which indicate that there is over-dispersion (i.e., greater variability) of the parameter alpha ($p<0.001$) in all models. Negative binomial models address the overdispersion of count outcomes by relaxing the assumption that the mean and variance of out count variable is equal (as is assumed in other Generalized Linear Models such as Poisson) (Long and Freese 2006).

In a fourth step, I compare the age at first birth of women with eating disorders by eating disorder type (anorexia, bulimia, and eating disorder-NOS) using Cox proportional hazard models. I also examine the influence of eating disorder severity and treatment on age at first birth by including age at first diagnosis, and eating disorder hospitalization. These models are stratified by birth year, meaning rather than assuming the baseline

hazard is the same for all individuals, each birth year is given its own calculated baseline hazard for fertility to address potential confounding cohort effects.

In a fifth step, I compare the parity of women with eating disorders by eating disorder type (anorexia, bulimia, and eating disorder-NOS) using negative binomial regression. I also examine the influence of eating disorder severity on parity by including age at first diagnosis and hospitalization. These models are adjusted for clustering by birth year to account for potential confounding cohort effects, most pertinent to parity analyses involving the incomplete fertility of younger cohorts.

Results

Table 3.1 presents descriptive statistics of our eating disorder sibling and general population samples. The sibling sample contains 503 sister pairs (1006 sisters total), and a general population sample of 1278 women without an eating disorder. Sisters without an eating disorder report slightly higher numbers of children (mean=0.62, *SD*=1.07) than their sister with an eating disorder (mean=0.34, *SD*=0.69). The general population sample had the highest average number of children of the three groups (mean=1.31, *SD*=1.75). Women with an eating disorder report older average ages at first birth of 25.14 years (*SD*=4.25), than their sisters (mean=24.78 years, *SD*=4.30) or the general population sample (mean=23.56 years, *SD*=3.81). Most notable are differences in suicide attempt/self-inflicted injury events between the eating disorder, sibling, and general population samples; 20.68 percent of women with an eating disorder were treated for a suicide attempt/self-inflicted injury before the birth of their first child, while only 3.38 percent of their sisters and 1.15 percent of the general population were treated for suicide attempt/self-inflicted injury.

Table 3.2 presents descriptive statistics of the full eating disorder analytic sample. Women with bulimia were more likely to have had a child (22.85 percent) than women with anorexia (17.86 percent) or eating disorder-NOS (18.33 percent). Women with anorexia had the highest average age at first birth (26.7 years, *SD*=4.62), compared to women diagnosed with bulimia (25.63 years, *SD*=4.40) or eating disorder-NOS (25.66 years, *SD*=5.82). All three forms of eating disorder had similar ages at first diagnosis (between 23-24 years); however, women with anorexia report higher rates of hospitalization for an eating disorder (68.36 percent report having been hospitalized at least once) compared to women diagnosed with bulimia (60.63 percent) and women diagnosed with eating disorder-NOS (50.76 percent).

Table 3.3 presents the Cox proportional hazard results examining the influence of eating disorder type on risk of first birth. Results are presented as hazard rate ratios (*HRR*s). Model 1 presents results of a sibling comparison, while Model 2 presents results of a general population comparison. These analyses are stratified by an eating disorder person identifier, which calculates a baseline hazard for each sibling pair (Model 1) or eating disorder general population match set (Model 2). Model 1 indicates that the hazard (i.e., risk) of first birth is lower for sisters with anorexia (*HRR*=0.37 [95 percent Confidence Interval=0.21-0.65], $p<0.001$) or eating disorder-NOS (*HRR*=0.60 [0.39-0.92], $p<0.05$) compared to sisters without an eating disorder. However, women with bulimia did not have significantly different risk of first birth compared to sisters without an eating disorder. Model 2 indicates that the hazard rate of first birth is significantly lower for all eating disorder types, with women diagnosed with anorexia having the lowest hazard (*HRR*=0.37 [0.25-0.56], $p<0.001$), compared to women diagnosed with

eating disorder-NOS (*HRR*=0.47 [0.34-0.65], $p<0.001$), or bulimia (*HRR*=0.56 [0.42-0.76], $p<0.001$) relative to the general population without an eating disorder.

Table 3.4 presents negative binomial regression results assessing the influence of eating disorder type on parity, or number of births. These results are presented as incidence rate ratios (*IRR*). Model 1 presents results of the sibling comparison, while Model 2 presents results of the general population comparison. These analyses are clustered by eating disorder person identifier. Model 1 indicates that women with anorexia and women with eating disorder-NOS have lower parity than their siblings without an eating disorder (*IRR*=0.56 [0.39-0.80], $p<0.001$ and *IRR*=0.57 [0.43-0.76], $p<0.001$ respectively), but that women with bulimia did not have significantly different parity. Model 2 indicates that the rate of birth of women with any eating disorder type is significantly lower relative to the general population without an eating disorder ($p<0.001$ for all eating disorder subgroups).

Table 3.5 presents the Cox proportional hazard results examining the influence of eating disorder type on risk of first birth of the full eating disorder analytic sample (n=1565). These results are stratified by birth year to correct for nonproportionality of birth year effects. These results indicate that, when controlling for age at eating disorder diagnosis, hospitalization for an eating disorder, suicide attempt/self-inflicted injury event, BMI, race/ethnicity, and household income (measured per $1,000 at the census block level), women with bulimia have increased risk of first birth compared to women with anorexia (*HRR*=1.38 [1.01-1.88], $p<0.05$). Women with eating disorder-NOS were not found to have significantly different risk of first birth compared to women with anorexia. Age at first eating disorder diagnosis is also significant, where a one-year

increase in age corresponds with a decreased hazard of first birth by 12 percent (*HRR*=0.88 [0.86-0.91], $p<0.05$).

Table 3.6 presents the negative binomial regression results assessing the influence of eating disorder type on number of births of the full eating disorder analytic sample. These results are adjusted for clustering by birth year to address potential confounding cohort effects. These results indicate that women with bulimia have a 41 percent increased expected number of births compared to women diagnosed with anorexia (*IRR*=1.41 [1.14-1.74], $p<0.001$), when covariates are held constant. Compared to women with anorexia, women with eating disorder-NOS did not have significantly different parity.

Discussion

This study utilized a sibling comparison design to assess the influence of eating disorder types on fertility trajectories. Analyses indicate that, compared to the general population, women with an eating disorder diagnosis (anorexia, bulimia, or eating disorder-NOS) experienced later ages at first birth, and lower total parity. However, while sisters diagnosed with anorexia nervosa or eating disorder-NOS *were* found to experience later ages at first birth, and lower total parity compared to their sibling, women with bulimia nervosa *were not* found to have significantly different fertility experiences than sisters without an eating disorder. These results indicate that women with diagnoses of anorexia nervosa or eating disorder-NOS may be experiencing true delay or disruption to their fertility due to their eating disorder, independent of their early life and familial experiences. The fertility experiences of women with bulimia, however, may be attributed to underlying epigenetic or early life experiences.

These results are supported in analyses comparing the fertility experiences of the full analytic sample of women diagnosed with an eating disorder: even when controlling for eating disorder experiences (e.g., age at first birth and hospitalization) women with bulimia were at higher risk of first birth (i.e., earlier ages of first birth), and higher overall parity than women with anorexia nervosa. Women diagnosed with eating disorder-NOS were found to have similar fertility experiences in comparison to women with anorexia.

These results are important given the limited research examining the fertility of women diagnosed with bulimia or eating disorder-NOS, and the dearth of research assessing *differences* in fertility experiences by eating disorder type. These results reconfirm the public health message that all forms of eating disorders have deleterious consequences for health and wellbeing, but that fertility interventions and screenings may be most beneficial for women with anorexia or eating disorder-NOS.

There are a several limitations of this study; although by stratifying the analyses by sibling pair, this study was able to make comparisons free from confounding factors that are shared by the sibling (e.g., familial environment, genetic similarity), there was no way to account for the potential statistical bias that may occur from the nonshared confounders between siblings (Frisell et al. 2012) beyond body mass index, being hospitalized for self harm or suicide attempt, and marital experiences. Some potential nonshared confounding factors of siblings include socioeconomic status in early adulthood (including personal earnings and education), differences in personality or preference, and additional health behaviors such as substance use. These nonshared confounding factors may explain why women with bulimia do not have significantly different fertility experiences than their closest aged sister. Although this limitation is

mitigated partially by the inclusion of a general population sample, where we do see significant differences in the fertility experiences of women with bulimia, future studies should attempt to account for additional contextual factors and outcomes commonly associated with eating disorders in order to better isolate eating disorders as a divergent life experience.

In addition, given the familial and genetic etiology of eating disorders (Campbell et al. 2011; Polivy and Herman 2002; Strober and Humphrey 1987; Strober et al. 2000), and that eating disorders are likely to go undiagnosed or untreated (Cachelin et al. 2001; Eisenberg et al. 2011; Hart et al. 2011), it is possible that the sister pairs are not truly discordant; the sibling identified as not having an eating disorder may simply have an eating disorder that has gone undiagnosed or was not captured in our sampling design (i.e., were treated by a small outpatient clinic). Descriptive statistics indicated that women with eating disorders *and* their sisters had reduced total fertility and delayed fertility in comparison to the general population. The similarity in fertility between sisters may be due to the fact that these sisters are not discordant. If these sisters are *not* discordant, that is, one sister has an undiagnosed or unidentified eating disorder, eating disorders may be more prevalent than has been previously acknowledged.

However, if the sisters *are* discordant and yet sisters *without* an eating disorder are *also* likely to experience reduced fertility, this may mean that the familial etiology of eating disorders in-and-of-itself may be contributing to reduced and/or delayed fertility. That is, the similarity in their reduced fertility is attributable to familial environmental or genetic factors. The similarity in the parity of sisters in this study is important given the contemporary trend toward reduced/delayed fertility among US females (Martin et al.

2015). Mental health conditions such as eating disorders, *and* their familial etiological factors, may be contributing to reduced/delayed fertility in the US. Etiological research indicates that parental support is protective against eating disorders (McVey et al. 2002). It is possible that, while discordant on the development of an eating disorder, both sisters are likely to experience reduced maternal or paternal involvement across the life course that may be contributing to their reduced fertility.

In addition, this study does not take into account fertility intentions. Differences in fertility experiences may be an artifact of differential desires for parenthood or family by eating disorder type, or among women with eating disorders in general. The eating disorder literature does indicate that women with eating disorders may have lower fertility intentions than women without eating disorders. For example, women with eating disorders are more likely to express unhappiness with being pregnant (Easter et al. 2011), and are more likely to induce abortion following a pregnancy (Linna et al. 2013). In a qualitative study, women with eating disorders expressed anxiety in early pregnancy due to body changes and weight gain, perceiving these changes as a loss of control over their body or weight (Taborelli et al. 2015). Women with eating disorders may have lower fertility intentions, potentially due to fears of weight gain, which may be translating to lower completed or delayed fertility.

Finally, this study excludes women who were diagnosed with an eating disorder after the birth of their first child. Average age of eating disorder onset is somewhere between 18-21 years depending on eating disorder type (Hudson et al. 2007), which is well into a woman's fertility. Eating disorders are common in women of reproductive age, and are therefore likely to be experienced *concurrently* with first and later

pregnancies (Cardwell 2013; Taborelli et al. 2015; Ward 2008). Future research should examine more extensively how eating disorder onset, treatment timing, and co-occurrence of eating disorders with fertility influence parity and fertility timing by eating disorder type.

Conclusion

Eating disorders are known to reduce female fertility through physical mechanisms, such as menstrual disruption (Cardwell 2013; James 2001; Stewart et al. 1990). However, little research has compared the full fertility experiences of women with eating disorders, in particular timing and parity, by eating disorder disease type. This study contributes to our knowledge of the consequences of eating disorders by examining the fertility experiences of women by eating disorder type, and the use of a discordant sibling-comparison design.

Results of this study indicate that women diagnosed with anorexia, bulimia, or eating disorder-NOS experience delayed and reduced total fertility in comparison to the general population, yet women diagnosed with bulimia did not experience significant differences in timing and parity in comparison to their closest aged sister without an eating disorder. This may mean that the causes of fertility delay or reduction in bulimia may be more attributable to familial environmental or genetic factors. In addition, women with bulimia were found to have significantly higher parity and experience lower ages at first birth than women with anorexia, while women with eating disorder-NOS had similar fertility experiences as women with anorexia. In short, women with anorexia or eating disorder-NOS may bet experiencing the greatest reductions and delays in fertility across

their lifespan, while women with bulimia may have fertility experiences that are more similar to the general population.

Table 3.1 Descriptive Statistics of Eating Disorder, Sibling, and General Population Samples

	Sibling with eating disorder (*n*=503)	Sibling without eating disorder (*n*=503)	General population matches without eating disorder (*n*=1,217)
	% or Mean (*SD*)	% or Mean (*SD*)	% or Mean (*SD*)
Independent Variable			
Diagnosed with ED			
Anorexia nervosa	25.65%	-	-
Bulimia nervosa	30.02%	-	-
Eating disorder-not otherwise specified	44.33%	-	-
Dependent Variable			
Number of children (Range 0-6)	0.34 (0.69)	0.62 (1.07)	1.31 (1.75)
Age at first birth (Range 16-38)	25.14 (4.25)	24.78 (4.30)	23.56 (3.81)
Covariates			
Race/Ethnicity			
Non-Hispanic white	90.06%	93.04%	92.36%
Hispanic	6.16%	3.98%	4.60%
Non-Hispanic other	3.78%	2.98%	3.04%
Marital status[a]			
Married	22.66%	35.59%	28.92%
Not married	77.34%	64.41%	71.08%
Body Mass Index[b] (Range 11.3-75.0)	21.68 (5.38)	22.05 (4.45)	22.28 (4.40)
Suicide attempt/self-inflicted injury Event[a]			
No	79.32%	96.62%	98.85%
Yes	20.68%	3.38%	1.15%

Notes: data come from the Utah Population Database (UPDB)
[a]Compares those married before the birth of first child (or age 50/age at last known in Utah for childless subjects), to those who were not married.
[b]Measured at earliest known time point beginning at age 12, before the birth of first child (or age 50/age at last known in Utah for childless subjects), using medical records, and height and weight information from the driver's license division.
[c]Compares those who have ever been treated for self-inflicted injury or suicide attempt before the birth of their first child (or age 50/age last known in Utah for childless subjects) to those who have never been treated.

Table 3.2 Descriptive Statistics of Full Eating Disorder Analytic Sample

	Women with Eating Disorders (*N*=1,565)		
	Anorexia nervosa (*n*=392)	Bulimia nervosa (*n*=442)	Eating disorder-not otherwise specified (*n*=731)
	% or Mean (*SD*)	% or Mean (*SD*)	% or Mean (*SD*)
Dependent Variable			
Fertility event			
Yes	17.86%	22.85%	18.33%
No	82.14%	77.15%	81.67%
Age at first birth (Range 16-42)	26.7 (4.62)	25.63 (4.40)	25.66 (5.82)
Number of Kids (Range 0-5)	0.28 (0.67)	0.33 (0.69)	0.27 (0.64)
Covariates			
Marital Status[a]			
Married	28.06%	25.57%	18.60%
Not married	71.94%	74.43%	81.40%
Suicide attempt/self-inflicted injury event[b]			
One or more event	19.90%	26.02%	18.60%
No event	80.10%	73.98%	81.40%
Race/ethnicity			
Non-Hispanic white	87.24%	85.97%	86.05%
Hispanic	9.18%	10.41%	8.21%
Non-Hispanic other	3.57%	3.62%	5.75%
Household income[c]	54,418 (20722)	53,916 (19340)	52,133 (20302)
Body Mass Index (BMI) (Range 11-75)[d]	19.58 (4.16)	22.98 (5.98)	23.10 (6.46)
Eating Disorder Severity Covariates			
Hospitalization for eating disorder[e]			
Never	31.63%	39.37%	49.25%
Once	36.22%	37.78%	41.59%
More than once	32.14%	22.85%	9.17%
Age at eating disorder diagnosis (Range 12-50)	23.44 (8.22)	23.52 (7.3)	23.17 (8.25)

Notes: Data come from the Utah Population Database (UPDB)

[a]Compares those married before the birth of first child (or age 50/age at last known in Utah for childless subjects), to those who were not married.

[b]Compares those who have ever been treated for self-inflicted injury or suicide attempt before the birth of their first child (or age 50/age last known in Utah for childless subjects) to those who have never been treated.

[c]Measured as median household income (dollars) at the census block level, measured before the birth of first child (or age 50/age last known in Utah for childless subjects).

[d]Measured at first eating disorder event based on the medical record. For those with missing BMI at eating disorder event, BMI was generated from height and weight information from driver's license issued nearest in age to event, but before the birth of first child or age 50/age at last known in Utah for childless subjects.

[e]Compares those who were not hospitalized for an eating disorder before the birth of their first child (or age 50/age last known in Utah for childless subjects), to those who were hospitalized once, to those who were hospitalized more than once.

Table 3.3 Cox Proportional Hazard Results (Hazard Rate Ratios): Influence of Eating Disorder on Risk of Age at First Birth

Age at First Birth	Model 1	Model 2
	Sibling sample	General population sample
Eating disorder[a]		
Anorexia nervosa	0.37***	0.37***
	(0.11)	(0.08)
Bulimia nervosa	0.73	0.56***
	(0.21)	(0.11)
Eating disorder-not otherwise specified	0.60*	0.47***
	(0.13)	(0.08)
Birth year[b]	1.17***	-
	(0.05)	-
Married[d]	3.98***	2.67***
	(1.18)	(0.27)
Body Mass Index (BMI)	0.94**	0.97*
	(0.03)	(0.01)
Suicide attempt/self-inflicted injury event[e]	0.81	1.49
	(0.30)	(0.36)
N	1006	1720

Notes: Data come from the Utah Population Database; hazard rate ratios reported; robust standard errors in parentheses. Models are stratified by eating disorder person identifier (EDpersonid). Supplementary analyses (not shown) including age at BMI measurement were run to ensure that BMI measurement differences were not significantly affecting results. Proportional hazards assumption was tested post estimation using Schoenfield residuals, and log-log plots of survival by eating disorder category.

*p<0.05 **p<0.01 ***p<0.001

[a]Reference group is no eating disorder

[b]Birth Year is excluded from general population model due to stratification of model by EDpersonid; given that a general population sample was drawn using a matched design, birth year matches perfectly by EDpersonid and its effect is absorbed into the baseline hazard. In supplementary analyses (not shown) of models not stratified by EDpersonid that include birth year, the main effects of eating disorder categories did not change

[c]Reference group is non-Hispanic white

[d]Reference group is unmarried before first birth (event) or right censor

[e]Reference group is no suicide attempt/self-inflicted injury event before first birth (event) or right censor

Table 3.4 Negative Binomial Regression Results (Incidence Rate Ratios): Influence of Eating Disorder on Number of Children

Number of Children	Model 1	Model 2
	Sibling sample	General population sample
Eating disorder[a]		
Anorexia nervosa	0.56***	0.21***
	(0.10)	(0.04)
Bulimia nervosa	0.96	0.34***
	(0.15)	(0.05)
Eating disorder-not otherwise specified	0.57***	0.21***
	(0.15)	(0.03)
Birth year	1.00	0.96
	(0.01)	(0.01)
Race/ethnicity[c]		
Hispanic	1.87**	2.89***
	(0.40)	(0.23)
Non-Hispanic other	0.80	0.99
	(0.30)	(0.24)
Married[d]	5.21***	2.86***
	(0.74)	(0.23)
Body Mass Index (BMI)	0.97*	0.98*
	(0.01)	(0.01)
Suicide attempt/self-inflicted injury event[e]	1.07	1.10
	(0.25)	(0.25)
N	1006	1720
Pseudo R-squared	0.125	0.084

Notes: Data come from the Utah Population Database; Incidence Rate Ratios reported; robust standard errors in parentheses. Models are estimated clustered by eating disorder person identifier (EDpersonid). Supplementary analyses (not shown) include age at BMI measurement to ensure that differences in measurement timing were not significantly biasing results.

*p<0.05 **p<0.01 ***p<0.001

[a]Reference group is no eating disorder

[c]Reference group is non-Hispanic white

[d]Reference group is unmarried before birth of first child, or age 50/age last known in Utah for childless subjects

[e]Reference group is no suicide attempt/self-inflicted injury event before birth of first child, or age 50/age last known in Utah for childless subjects

Table 3.5 Cox Proportional Hazard Results (Hazard Rate Ratios): Influence of Eating Disorder Type on Risk of Age at First Birth

Age at First Birth	Model 1
Eating disorder[a]	
Bulimia nervosa	1.38*
	(0.22)
Eating disorder-not otherwise specified	1.23
	(0.19)
Household income[b]	0.99**
	(0.00)
Race/ethnicity[c]	
Hispanic	1.23*
	(0.22)
Non-Hispanic other	0.92
	(0.19)
Married[d]	3.89**
	(0.51)
Body Mass Index (BMI)	0.99
	(0.01)
Suicide attempt/self-inflicted injury event[e]	0.77
	(0.13)
Eating disorder hospitalization[f]	
Hospitalized once	1.18
	(0.15)
Hospitalized more than once	0.56
	(0.12)
Age at first eating disorder diagnosis	0.88***
	(0.13)
N	1565

Notes: Data come from the Utah Population Database; hazard rate ratios reported; robust standard errors in parentheses. Models are stratified by birth year to address potential cohort effects. Supplementary analyses (not shown) including age at BMI measurement were run to ensure that BMI measurement differences were not significantly biasing results. In addition, the proportional hazard assumption was tested based on Schoenfield residuals; based on this global test, hazard results are not proportional. However, log-log plots (not shown) of survival by eating disorder category indicate that eating disorder subtypes hazard results are proportional.

*p<0.05 **p<0.01 ***p<0.001

[a]Reference group is anorexia nervosa

[b]Median household income at census block measured in dollars per thousand

[c]Reference group is non-Hispanic white

[d]Reference group is unmarried before first birth (event) or right censor

[e]Reference group is no Suicide attempt/self-inflicted injury event before first birth (event) or right censor

[f]reference group is no-hospitalization event before first birth (event) or right censor

Table 3.6 Negative Binomial Regression Results (Incidence Rate Ratios): Influence of Eating Disorder Type on Total Number of Children

Number of Children	Model 1
Eating Disorder[a]	
Bulimia nervosa	1.41***
	(0.15)
Eating disorder-not otherwise specified	1.13
	(0.17)
Median income[b]	0.99**
	(0.00)
Race/ethnicity[c]	
Hispanic	1.40**
	(0.18)
Non-Hispanic other	0.00
	(0.17)
Married[d]	5.21***
	(0.69)
Body Mass Index (BMI)	0.98*
	(0.01)
Suicide attempt/self-inflicted injury event[e]	0.84
	(0.15)
Eating disorder hospitalization[f]	
Hospitalized once	1.05
	(0.14)
Hospitalized more than once	0.63*
	(0.13)
Age at first eating disorder diagnosis	0.94***
	(0.01)
N	1565
Pseudo R-Squared	0.10

Notes: Data come from the Utah Population Database; Incidence Rate Ratios reported; robust standard errors in parentheses. Models are clustered by birth year to address potential cohort effects. Supplementary analyses (not shown) including age at BMI measurement were run to ensure that BMI measurement differences were not significantly biasing the results.

*$p<0.05$ **$p<0.01$ ***$p<0.001$

[a]Reference group is Anorexia nervosa

[b]Median household income at census block level measured in dollars per thousand

[c]Reference group is non-Hispanic white

[d]Reference group is unmarried before first birth (or age 50/last known in Utah for childless subjects)

[e]Reference group is no Suicide attempt/self-inflicted injury event before first birth (or age 50/last known in Utah for childless subjects)

[f]Reference group is no-hospitalization event before first birth (or age 50/last known in Utah for childless subjects)

CHAPTER 4

YOUNG WOMEN WITH EATING DISORDERS OR DISORDERED EATING BEHAVIORS: DELINQUENCY, RISKY SEXUAL BEHAVIORS, AND NUMBER OF CHILDREN IN EARLY ADULTHOOD[7]

Introduction

Although research has shown that eating disorders during adolescence can have lasting effects on health, very few studies have explored the role that eating disorders or disordered eating behaviors play on the transition to adulthood. For example, it is well established that women with eating disorders are more likely to experience infertility and difficulty becoming pregnant (James 2001; Linna et al. 2013; Stewart et al. 1990); however, it is unclear how eating disorders may be influencing the transition to, and experiences of, parenthood more broadly, such as age at entry into parenthood, number of children, and birth timing. Examining the fertility timing and overall parity of women with eating disorders is important given recent evidence linking eating disorders with higher rates of unintended pregnancy (Bulik et al. 2010; Easter et al. 2011).

Using a nationally representative sample from the National Study of Adolescent and Young Adult Health (Add Health), we seek to empirically account for how eating disorders (EDs) or disordered eating behaviors (DEBs) in early life may influence women's childbearing in early adulthood. The primary research objective of this study is

[7] Co-authored by Claudia Geist. *Socius: Sociological Research for a Dynamic World* has accepted a version of this manuscript for publication. Author retains the right for dissertation use.

to determine whether EDs or DEBs influence the number of children young women have by the time they reach early adulthood. We also test two potential mediating factors that may explain the relationship between eating disorders or disordered eating behavior and parity in early adulthood: adolescent delinquency and sexual risk taking. Our approach is novel in that we apply a sociological life course framework, and utilize a nationally representative sample, to fertility research on women with eating disorders, which has generally been studied from a biomedical perspective utilizing clinical samples.

Background

Eating Disorders and Disordered Eating Behaviors

EDs, defined here as the range of psychological disorders characterized by abnormal or disturbed eating habits identified by the Diagnostic Statistical Manual of Mental Disorders (DSM-5) (APA 2013), often begin in adolescence, with an average age of diagnosis at 17-18 years (Fairburn and Harrison 2003). Swanson et al. (2011) estimate that approximately 2.7 per 100,000 adolescents between the ages of 13-18 in the United States have an ED. Since many EDs are not diagnosed, these estimates are likely under-represented measures of true prevalence. EDs are often chronic conditions that persist into adulthood, with full recovery only occurring for approximately half of adolescent patients (Yager and Andersen 2005).

DEBs are the behavioral symptoms of EDs, such as purging, binge eating, and other compensatory behaviors (e.g., fasting or taking laxatives), and are much more common than diagnosed EDs (Stephen et al. 2014). DEBs are likely to develop during adolescence (Neumark-Sztainer et al. 2011; Neumark-Sztainer et al. 2012); longitudinal studies indicate that DEBs increase from early to late adolescence, meaning an

adolescent's risk for developing and engaging in DEBs increases across adolescence (Neumark-Sztainer et al. 2011). The high prevalence of DEBs among adolescents and their correlation with adverse physical health and psychosocial outcomes (such as depression and weight gain) illustrates that DEBs have long lasting impacts on wellbeing (Neumark-Sztainer et al. 2011; Neumark-Sztainer et al. 2012; Stephen et al. 2014). Although DEBs have been traditionally utilized as proxy measures of EDs, DEBs are arguably clinically relevant independent of whether they evolve into fully-fledged EDs based on diagnostic criteria.

We rely on a life course framework, which suggests that early life experiences influence later life outcomes (Elder 1998; Mortimer and Shanahan 2003). EDs often occur during the life phase called the transition to adulthood. During the transition, most adolescents have been dependent on parents but with age are transitioning into independence both financially and emotionally (Jekielek and Brown 2005). Some adolescents or young adults engage in behaviors or experience setbacks that place the young adult on a disadvantaged trajectory, leading to cumulative disadvantages later in life (Jekielek and Brown 2005). Adolescence and the transition to adulthood is often described as a time when individuals engage in risk-taking behaviors (Steinberg and Silverberg 1986), such as DEBs (Neumark-Sztainer et al. 2011). Given that adolescents who engage in risky behaviors are more likely to take subsequent, or additional risks, for example, adolescents who engage in substance use are more likely to engage in risky sexual behaviors throughout the transition of adulthood (Tapert et al. 2001), the presence of EDs in adolescence or young adulthood may indicate a constellation of deviant or risk-taking behaviors.

Events, experiences, or sets of behaviors that result in a significant change to the life or health trajectory of an individual are referred to as "turning points" (Wheaton and Gotlib 1997). EDs and related risk-taking behaviors may act as a turning point, setting an individual on a different life path, thereby resulting in differential successes during the transition to adulthood. We seek to assess whether EDs disrupt or alter the transition to adulthood by affecting the fertility experiences of women. In order to assess EDs as a particular divergent experience, we need to account for the known etiology, associated deviant or risk-taking behaviors, and consequences of adolescent EDs. Examining EDs from a life course perspective is important practically because it improves our understanding of how EDs or DEB may influence parenthood in early adulthood. This work is also important theoretically because it attempts to understand the validity of life course theory and life course concepts as they apply to EDs.

Underlying Causes and Risk Factors

Gender is one of the most important risk factors predicting the development of EDs and DEBs. Females have been consistently shown to have higher lifetime prevalence of EDs than males, and are more likely to engage in DEBs (Hudson et al. 2007; Santos et al. 2007; Stephen et al. 2014). It is well established that females are more prone to societal pressures to maintain or achieve thinness (Grogan 2007). This study explores the relationship between EDs, DEBs, and fertility among a female-only sample for two main reasons. First, the meanings associated with EDs and DEBs are vastly different for men and women. For example, although both men and women with EDs or DEBs exhibit higher perfectionism (Bardone-Cone et al. 2007), characteristics of "perfection" differ by gender. Females with EDs or DEBs may place more value onto their physical appearance

than males with EDs or DEBs due to female-specific social pressures for thinness (Grogan 2007). Indeed, males with EDs do not score as high in bodily dissatisfaction and drive for thinness (Stanford and Lemberg 2012). Second, measuring fertility among men is more difficult, and because of their greater likelihood to be nonresident with their child(ren) (Sorensen 1997), the implication of early adulthood fertility differs substantially between the two groups. As a result, this study focuses exclusively on the experiences of women.

While earlier research suggested that lesbians have a more positive body image and might be less likely to engage in disordered eating (Morrison, Morrison, and Sager 2004; Striegel-Moore, Tucker, and Hsu 1990), recent studies indicate that sexual minority women are at higher risk for both purging and diet pill use (Austin et al. 2013). This suggests that, in addition to experiencing minority stress, they are not exempt from heterosexist body standards for women (Watson et al. 2015), so it is important to explicitly include this factor in studies of young adults.

The home environment has an important influence on the development of EDs and/or DEBs (Jacobi et al. 2004); in a systematic review of the causes of EDs, Polivy and Herman (2002) identify negative family environments and lack of familial support during early childhood as major risk factors for EDs. EDs are often understood as coping mechanisms for the perceived lack of control or support in the home environment (Polivy and Herman 2002; Wagener and Much 2010). Many societies emphasize culturally idealized thinness, and individuals with EDs are aware of the importance of this ideal, internalize it, and perceive pressure from the media and their peers to be thin (Levine and Murnen 2009). Although researchers have grappled over how to change predominating

cultural perspectives idealizing thinness (Levine and Murnen 2009), increased positive social support from peers and other adults is protective against disordered eating and the development of EDs, thereby mitigating some of these unhealthy cultural pressures (Limbert 2010; McVey et al. 2003).

In addition, race/ethnicity and socioeconoemic status (SES) have been found to influence female drive to thinness, with research indicating that Asian American and white women, particularly those from high SES backgrounds, are more driven to be "thin" than African Americans or Hispanics (Boyd et al. 2011). Therefore, it is commonly stated that racial and ethnic, and class-specific, identity group relations and dynamics shape female susceptibility to the thinness ideal of mainstream culture, which may be translating to differences in type-specific ED prevalence manifestation (Boyd et al. 2011). For example, white females and those from higher SES backgrounds experience higher prevalence of anorexia nervosa (Swanson et al. 2011), while African Americans and Latinos are found to have higher prevalence of bulimia nervosa (Franko et al. 2007; Marques et al. 2011).

Finally, evidence suggests that EDs are the result of genetic and epigenetic processes, that is, the interaction between the environment and genes (Campbell et al. 2011; Goodman et al. 2014; Strober et al. 2000). When individuals' genetic predispositions interact with environments rich in potential risk factors, such as a stressful home environment, their likelihood of developing an ED or DEB increases. Indeed, there is a high risk of familial transference associated with EDs, where families "pass" EDs through the family environment, usually from parent to child (Polivy and Herman 2002; Strober et al. 2000). For example, mothers with EDs may have higher

expectations for their daughter's thinness, and may be less positive about their daughter's "attractiveness," prompting the child to engage in DEBs (Polivy and Herman 2002). This makes understanding fertility behaviors of women with EDs and DEBs all the more important.

Fertility Consequences

There are two different approaches to examining the link eating disorders and disordered eating behaviors and fertility outcomes. *Clinical studies* indicate that women with prolonged or severe eating disorder may have difficulty getting pregnant as a result of physical health complications (James 2001; Linna et al. 2013; Stewart et al. 1990). It is well established that eating disorders and disordered eating behaviors are associated with long-lasting mental and physical health consequences (Berkman et al. 2007; Hudson et al. 2007; Nicholls et al. 2011; Norris et al. 2012; Swanson et al. 2011). More severe and prolonged EDs and DEBs have the most severe and longest-lasting effects on physical and mental health (Yager and Andersen 2005). The physical health consequences of eating disorders, low-body-weight-related menstruation disruption in particular, have been linked to lifetime fertility problems (Freizinger et al. 2010; Stewart et al. 1990; Stewart 1992), with women with eating disorders expressing difficulty becoming pregnant and/or experiencing longer times to conception (Easter et al. 2011). In terms of the influence of eating disorders on parity, or number of children born, Linna et al. (2013) found that women seeking treatment for eating disorders were more likely to be childless than the control group in an observational study utilizing a clinical sample. Based on this body of medical research, we might hypothesize that women with adolescent eating disorders or disordered eating behaviors will have fewer children in early adulthood.

However, recent research indicates that women with EDs are at greater risk of experiencing unplanned pregnancy, particularly those studies utilizing broader community samples (Bulik et al. 2010; Easter et al. 2011). We build on this research by proposing an alternative sociological perspective that may provide insight into recent findings linking EDs and unplanned pregnancy.

Deviant Behavior and Risky Sexual Behaviors

Sociological research suggests an alternative understanding of the link between EDs/DEBs in adolescence or young adulthood and early adult fertility. Adolescent decision-making theory proposes that, while adolescents are able to assess the risk, benefit, and consequences of a particular decision or behavior, adolescents or young adults see occasional or experimental involvement in health-threatening activities as less dangerous than do adults (Cohn et al. 1995), and may also overestimate their ability to recognize and avoid dangerous situations or behaviors (Cohn et al. 1995). Adolescents or young adults with low self-esteem or negative self-image may be even less likely to appropriately understand or respond to the risk or consequences of, and more likely to engage in, risky behaviors (Smith, Gerrard, and Gibbons 1997; Wheeler 2010).

Similarly, DEBs have been conceptualized as a form of internally directed deviance, resulting from negative self-image and low self-esteem (Sischo et al. 2006). EDs and DEBs have been associated with other forms of externalized deviance and risk behaviors; for example, EDs and DEBs in adolescence are also associated with higher levels of delinquency and substance use (Piran and Robinson 2011; Stephen et al. 2014; Striegel-Moore et al. 2003); such risky behaviors are also commonly associated with a constellation of additional risk behaviors. Important to this study is their association with

risky sexual behavior, including early age of first sexual encounter and higher numbers of sexual partners, and early or unintended pregnancy (Naimi et al. 2003; Pugh et al. 1990; Yamaguchi and Kandel 1987).

Indeed, there is some evidence to conclude that young adults diagnosed with one or more psychiatric disorders, including eating disorders, are more likely to engage in risky sexual intercourse (noncondom use, higher number of partners) and have sexual intercourse at an early age (Ramrakha et al. 2000; Shrier et al. 2001). Young women with ED or DEB may be particularly prone to risky sexual behavior, including earlier ages of first sexual intercourse and higher number of sexual partners, due to their compromised self esteem (Fisher et al. 1991). In addition, women with EDs or DEBs may also be less likely to use effective forms of contraception due to their assumption that they may be infertile and/or a reduced perception of risk (Bulik et al. 2010; Downs et al. 2004), resulting in higher rates of unplanned pregnancy (Bulik et al. 2010; Easter et al. 2011).

Because unintended pregnancy is a risk factor for subsequent, unintended pregnancies (Kuroki et al. 2008), women with ED or DEB may not only be at risk of early entry into parenthood, but of experiencing multiple births at a younger age than their unafflicted peers. Our study builds on research that has shown that women with EDs are at greater risk of experiencing unplanned pregnancy, particularly those studies utilizing broader community samples (Bulik et al. 2010; Easter et al. 2011). Based on the sociological literature, we expect EDs and DEBs to be associated with higher parity in early adulthood.

Clinical versus Nonclinical Samples

Prior research examining the influence of eating disorders on fertility has relied on clinical sampling or clinical measurement (i.e., diagnosis) of an ED. To be diagnosed with an ED and included in a clinical study, an individual must have access and the desire to seek the help of a medical professional. As Cohen and Cohen (1984) note, clinical samples are therefore biased toward cases of long duration or greater severity, and/or capture individuals actively seeking treatment for an illness or condition, thereby limiting the generalizability of clinical findings to the broader population. This reduces the generalizability of prior clinical studies that examine the influence of EDs on fertility experiences to diverse populations.

Conversely, it can be difficult to examine the influence of EDs on fertility within community or national samples, which better represent the general population, given that there can be few individuals who are diagnosed with EDs within these samples. This has led to the use of proxy measures of EDs, including DEBs, in place of clinical diagnosis within nonclinical studies (Stephen et al. 2014; Tabler and Utz 2015). Asking respondents about DEBs that are indicative of eating disorders in community or population based surveys is an alternative way of assessing EDs that moves beyond utilization of medical services, and may be able to capture individuals who are underrepresented in samples of individuals who are diagnosed and/or actively seeking treatment for an ED.

Current Study

In the current study, we examine whether EDs and DEBs are additional risk factors for higher parity in early adulthood, while taking into account other individual

characteristics and behaviors, including delinquency and risky sexual behaviors. This study utilizes a nationally representative, longitudinal sample of adolescents and young adults in the United States (US), and examines a combined measure of self-reported diagnosis and disordered eating behavior. The unique sampling design and ED measurement techniques in this study allows for greater generalizability to the US population than prior work examining the influence of EDs on fertility experiences. This study is important because it has the potential to expand our current understanding of the transition to parenthood of women with adolescent EDs and DEBs, beyond strictly biomedical considerations. Indeed, most fertility studies sample women with anorexia nervosa and/or women who are trying to get pregnant, and are therefore unrepresentative of the overall fertility experiences of women with EDs or DEBs. This study provides a sociological approach to the topic and broadens our understanding of the parenthood experiences of women with EDs or DEBs.

Methods

Data

The data used in this study come from the National Study of Adolescent and Young Adult Health, or Add Health, collected by the Carolina Population Center from 1994-2008 (Harris 2011). Add Health follows the same cohort of randomly selected youth from adolescence to young adulthood, collecting data at four different time periods. They used a stratified sampling design (i.e., children were sampled within selected schools). Wave I was collected between 1994-95, when the cohort was aged 11 to 18; Wave II was repeated in 1996; Wave III between 2001-02; and Wave IV was collected in 2008 when the cohort was approximately aged 24 to 32. The current study uses data from

Waves I, III, and IV. Specifically, the early life contexts and demographic information were selected from Wave I. Information about ED diagnoses and behaviors were only available at Wave III. Finally, Wave IV provides measures of socioeconomic independence, health, marital status, and entry into parenthood during early adulthood. Wave II was excluded because it did not add additional information relevant to these analyses.

Sample

Of the 10,480 women in the full data set, approximately 20 percent (*n*=2,128) of respondents were lost by attrition between Waves I and IV. An additional 3,008 individuals had missing values on key variables. Finally, an additional 307 were dropped due to missing longitudinal sample weights. This left a final analytic sample of 5,037 female respondents.[8] Table 4.1 presents the descriptive profile of the estimation sample, which was generated using Wave IV longitudinal sample weights for individuals who have responded to Waves I, III, and IV (Chen and Chantala 2014).

Measures

Dependent Variable

Parity was measured in Wave IV, when participants were in early adulthood, with the average age of 28 years for respondents (range=24-33). The national average age of first birth for women in the US is approximately 26 years (Mathews et al. 2009). We use the measure *Parenthood*, a categorical measure of number of children, which compares those with no children to those with one or two children, and to those with three or more.

[8] Supplementary analyses show that the analytic sample is somewhat different from the full sample. Most notably, adolescents who were Hispanic were significantly more prone to be excluded from the analytic sample.

We have selected this measurement of parity for three reasons; first, given prior research indicating that women with eating disorders are more likely to be childless than unafflicted peers (Linna et al. 2013), we selected childlessness as our baseline outcome. In addition, research indicates that the perceived benefits or burdens of having children are different for women who are childless compared to women who have one or two children (Callan 1986). For this reason, we selected women with one or two children as a comparison group. Finally, given that the average completed fertility of women in the US is approximately two children (Martin et al. 2015), we grouped women with three or more children as a final comparison group because they represent a subsample of women who have higher than average fertility.

Primary Independent Variable

Survey questions related to EDs and DEBs were only asked in Wave III. Self-identified ED diagnosis was assessed with a single yes/no question, “have you *ever* been diagnosed with an eating disorder?” We further identified individuals participating in unhealthy compensatory behaviors directed at maintaining or losing weight. Respondents were asked, “During the past seven days what did you do to keep from gaining weight?” Individuals who reported behaviors, "made your-self vomit, fasted or skipped meals, took laxatives, took diet pills, or diuretics,” were coded as having DEBs. We also identified individuals with binge eating symptoms. We included those who reported having “eaten so much in a short period of time that [they] would have been embarrassed if others had seen them do it, in the past seven days” as having a DEB. The measure labeled *ED or DEB* combines individuals who self-identify as having been diagnosed with an ED with those who engage in unhealthy weight-related compensatory behaviors, and/or exhibit

binge eating symptoms.

Covariates

Delinquency and Risky Sexual Behaviors

In this study, we examine whether risk behaviors commonly associated with eating disorders, including delinquent and risky sexual behaviors, may mediate the relationship between EDs or DEBs and parity. *Delinquency* was measured at Wave III, and is a composite score of 12 items. Questions evaluated how often in the past 12 months the individual engaged in delinquent behavior. Examples include "How often did you sell marijuana or other drugs?" and "How often did you steal something worth more than $50?" Potential responses include 0 "never," 1 "one or two times," 2 "three or four times," and 3 "five or more times." Scores can range from 0-36, with higher scores indicating more delinquent behaviors (α=0.710). A full list of Add Health measures and items can be found in the Appendix.

Three variables provide information on risky sexual behaviors. Two were measured at Wave IV. *Age at first vaginal sex* is measured in years (range=11-30).[9] *Number of sexual partners* represents a self-reported, estimated count of total number of sexual partners (range=1-100). *Contraceptive use* was measured at Wave III, and is a categorical measure of contraceptive type, comparing those using no birth control in the past 12 months, to those using at least one effective forms of birth control (including the pill, birth control implant, injection or shot, or diaphragm), and to those using ineffective forms of birth control (including natural planning techniques, and emergency contraception).

[9] Individuals who have not had vaginal sex were excluded from our analyses.

Additional Covariates

In an attempt to better isolate the association between EDs and DEBs and parenthood, we attempt to account for potential confounders that are associated with EDs/DEBs and parenthood. Thus, this study includes covariates that describe their early life context (in this case, early adolescence), the demographic characteristics of the individual, as well as the health and socioeconomic characteristics of the individual in early adulthood.

Two variables provide information on the individual's early life context. These variables were measured at Wave I, when participants were approximately 11-18 years of age. Respondents were asked to choose from six potential categories to specify the highest educational level of their mother (*Mother's Education*). Potential responses include "less than high school" up to "post baccalaureate degree". Respondents were also able to specify if they were unsure of their mother's highest level of education. The *Protective Environment Scale* captured the supportiveness of the child's social network using eight Likert-scale items that measured perceived social support from persons or groups in the child's social network. For example, respondents were asked, "How much do you feel that adults care about you?" 1 "Not at all" 2 "very little" 3 "somewhat" 4 "quite a bit" 5 "very much." Other questions assessed the perceived support from teachers, parents, friends, and family members. Based on the sum of all 8 items, scores ranged from 8-40, with higher scores representing more supportive social environments. The scale was found to have sufficient internal consistency (α=0.782).

Race/Ethnicity is a self-identified measure of an individual's race or ethnicity, measured at Wave I. Potential categories include "Hispanic," "Non-Hispanic white,"

"Non-Hispanic black," "Non-Hispanic Asian," and "Non-Hispanic other." We selected these categories due to sample size limitations; more detailed racial and ethnic groups were too small for meaningful comparisons by eating disorder or disordered eating behavior. *Age*, measured in years, was calculated by subtracting birth date from the survey date (of Wave IV). *Sexual Orientation*[10] was measured at Wave IV. This dichotomous variable compares those who identify as being entirely heterosexual (i.e., are exclusively attracted to individuals of the "other" sex) to individuals specifying having any level of same-sex attraction.

Health was measured at Wave IV, when participants were in early adulthood. *Mental Health* was measured by a version of the Center for Disease Epidemiology-Depression scale (CES-D) (Radloff 1977). The CES-D is a composite score of ten items indicating the presence of depressive symptoms such as, "You could not shake off the blues, even with the help from family and friends, in the past seven days." Possible values of the combined ten-item scale ranged from 0 to 30, with higher scores indicating higher levels of depressive symptoms (α=0.83). See the Appendix for exact items. *General Health* was measured using self-reported global health. Respondents were asked, "How is your general health?" Responses fall on a 5-point Likert-scale, ranging from 0 "poor" to 5 "excellent." Body Mass Index (BMI) was calculated by first using the height and weight data measured by the interviewer at Wave IV. Self-reported height and weight was used if the respondent had missing measured data (0.1 percent of original Add

[10] Although the focus on our paper is women's fertility patterns, there is also evidence that sexual minority women have a higher prevalence of sexual risk behaviors. Since their partners cannot be assumed to be all female, sexual minorities may also be at higher risk of pregnancy (see Mojola, Sanyu A. and Bethany Everett. 2012. "STD and HIV Risk Factors among US Young Adults: Variations by Gender, Race, Ethnicity and Sexual Orientation." *Perspectives on Sexual and Reproductive Health* 44(2):125-33).

Health sample).

In addition, we control for the education of respondents at Wave IV. *Educational Attainment* is a measure of years of completed education. Using the International Standard Classification of Education (ISCED), categorical responses to the question "what is the highest level of education that you have achieved to date" (i.e., completed high school, some college, completed master's degree) were transformed into years of completed school.[11] Finally, we controlled for the marital status of the respondent in early adulthood (Wave IV). *Married* is a dichotomous measure of having been married at least once.

Analytic Plan

In a first step, we present the unadjusted group differences between those with and without EDs/DEBs in our dependent and selected independent variables. In a second step, we estimate a series of nested multinomial logistic regression models. In a third step, we presented predicted probabilities to assess the substantive impact of EDs and DEBs on fertility patterns. All statistic results are based on sample estimates generated using Wave IV longitudinal sample weights (for individuals who responded to Waves I, III, and IV) based on guidelines for analyzing Add Health data (see Chen and Chantala 2014).

Results

About 3.9 percent of the sample reported an eating disorder (ED) diagnosis based on sample estimations. A higher proportion of individuals, 22.3 percent, reported

[11] Supplemental analyses using the rank-ordered categories of educational levels did not yield significantly different results.

engaging in at least one DEB. Of females, 23.5 percent reported having been diagnosed with an ED or engaging in at least one DEB (Table 4.1). Table 4.2 presents group differences that indicate initial support for our hypothesis as women with an ED or DEB were more likely to have had three or more children by early adulthood than those without and ED or DEB ($p<0.001$, based on Chi-squared tests). In addition, women with an ED or DEB report significantly higher levels of adolescent delinquency ($p<0.001$), younger ages at first vaginal sex ($p<0.001$), and higher numbers of sexual partners ($p<0.001$) than their unafflicted peers. Contraceptive use was not correlated with ED or DEB. We also see that identifying as nonheterosexual was positively correlated with identifying as having an ED or DEB ($p<0.001$). Respondents with an ED or DEB also reported lower general health ($p<0.001$), higher depression scores ($p<0.001$), and higher BMI in adulthood ($p<0.001$). In addition, women with ED or DEB experience lower social support in adolescence ($p<0.001$).

In a next step, we test whether this difference in fertility patterns remains once we estimate multivariate models (Table 4.3). We first present a model that includes eating disorder or disordered eating behavior and the full set of demographic controls, followed by a model that also includes delinquency and sexual risk taking.

In a next step, we test whether this difference in fertility patterns remains once we estimate multivariate models (Table 4.3). We first present a model that includes ED or DEB and the full set of demographic controls, followed by a model that also includes delinquency and sexual risk-taking behaviors. In Model 1, we compare those without children to those who have one or two children in early adulthood (Model 1, column 1), and to women who have three or more children (Model 1, column 2). The individual

controls have the expected effects: older individuals, and those who are already married, have greater relative risk to have children compared to having no children. Compared to non-Hispanic whites, African American women are more likely to have children in early adulthood, and higher levels of education (both respondents' and mother's) are associated with reduced risk of parenthood (at both levels). We find that sexual minority women are less likely to already have children compared to those who do not indicate any same sex attraction.

These results provide us with partial support for our hypothesis that young women with an ED or DEB had a higher relative risk for early transition to parenthood. While having an ED or DEB does not affect the relative risk of having one or two versus remaining childless, there is evidence that ED/DEB women have a greater relative risk of having three or more children compared to being childless in early adulthood ($p<0.05$).

In Model 2, we further include delinquency and risky sexual behaviors, a set of theoretically relevant factors that potentially mediate the relationship between ED or DEB and parity. Similar to Model 1, Model 2 compares those without children to those who have one or two children in early adulthood (Model 2, column 1), and to women who have three or more children (Model 2, column 2). Contraceptive use, delinquency, and number of sexual partners did not have direct effects on the relative risk of having one or two versus remaining childless, or having three or more children versus remaining childless. Age at first vaginal sex has a significant effect, where for each one year delay in age at first vaginal sex, women's relative risk of having one or two children versus remaining childless is reduced by 14 percent ($p<0.001$), and their relative risk of having three or more children versus remaining childless is reduced by 23 percent ($p<0.001$).

However, even when we include these potential explanatory variables, ED or DEB remains a significant predictor of parity, where women with ED or DEB have an increased relative risk of having three or more children compared to being childless in early adulthood by 46 percent ($p<0.05$).

We also explored additional models that compared other models with different combinations of delinquency and sexual risk taking and models presented here provide the best fit (full set of models available upon request). It is important to note that in supplementary analyses (not shown) where we excluded those engaging in DEBs, EDs remained a significant positive predictor of having three or more children in early adulthood.

Relative risk ratios do not take into account the underlying probabilities, so large differences reported in regression tables may not translate in substantive differences. In a third analytic step, we estimated probabilities for women with and without ED and DEB, with all other characteristics held at the overall sample mean. Overall, the probability of having three or more children is relatively small (<10 percent), which is not surprising since our oldest respondents are only 33 years old. However, we do find that those with ED or DEB have a significantly greater probability to report three or more children than those without (see Figure 4.1) ($p<0.05$).

Discussion

This study expands existing research on EDs and DEBs beyond health domains by using a nationally representative, community-based survey of adolescents to explore whether EDs or DEBs had an effect on parenthood in early adulthood. Despite previous research linking infertility and compromised reproductive health outcomes among

women with EDs (Linna et al. 2013), our findings indicate that adolescent women who experienced EDs or DEBs in early life were more likely to have three or more children by early adulthood. This findings holds even when we take into account adolescent risk taking and sexual behavior. This suggests that DEBs and particular diagnosed EDs have an independent effect on the odds of entering parenthood early, net of behaviors that increase the risk of early parenthood.

Our finding that women with EDs or DEBs are more likely to have three or more children in early adulthood indicates that women with EDs and DEBs may be entering motherhood at an increased tempo compared to their peers, particularly when we consider contemporary trends toward reduced or delayed fertility among US females (Martin et al. 2015); the US has seen a steady decline in births to women under the age of thirty, and an increase in birth rates among women aged 30-39 (Martin et al. 2015). This finding has important practical implications. Becoming pregnant while engaging in DEB places both mother and child at risk, and, given the genetic and familial nature of EDs, the children successfully born to women with EDs are in-and-of-themselves at greater risk of engaging in unhealthy eating behaviors (Pike and Rodin 1991; Strober et al. 2000).

Women with EDs or DEBs may be making different fertility decisions that move beyond sexual risk taking. Due to survey limitations, we were unable to account for adolescent fertility intentions, but differences in fertility intentions between women with or without EDs or DEBs may help explain their differences in completed fertility. Additional research on the attitudes of women with EDs or DEBs toward parenting, their birth timing, and lifetime parity is thus arguably important, not only for elucidating the

potential health risks of these women, but those posed to their children.

Early childbearing is known to lower the overall educational attainment of young women (Klepinger, Lundberg, and Plotnick 1995). Raising multiple children in early adulthood may place women with EDs or DEBs at cumulative risk for financial and emotional distress, given previous results indicating that young women who experience EDs or DEBs face barriers in achieving socioeconomic independence in early adulthood (Tabler and Utz 2015), and higher rates of depression and anxiety (Hudson et al. 2007; Santos et al. 2007). Disadvantaged women have been found to experience heightened insecurity and fears surrounding whether or not they may be able to achieve motherhood, driving them to pursue it at early ages (Edin and Kefalas 2005). Similarly, becoming pregnant may be more highly valued by women with EDs or DEBs because they fear they might not have the opportunity to achieve a successful pregnancy (or pregnancies), given the link between low self-esteem and unhealthy eating behaviors (Martyn-Nemeth et al. 2009). Both qualitative and quantitative research on the meaning of motherhood among women with EDs would enrich our understanding of the family formation intentions of women with EDs and DEBs.

Documenting the life course implications of adolescent ED and DEB is important from a public health perspective, but it also points to sociological implications. Beyond deviant behavior and sexual risk taking, EDs shape young women's entry into adulthood, and additional research needs to understand the gendered notion of the body, the striving for perfection, and the motivations for early entry into parenthood among women with EDs. Our research establishes that adolescent experiences, EDs and DEBs in particular, have life course altering potential.

There are several limitations of this study. Due to data limitations, we were unable to distinguish types of EDs or measure the severity or duration of reported DEBs. We expect that those with lifetime diagnoses or prolonged untreated behaviors would be more likely to experience fertility complications due to the physical health complications commonly associated with severe and ongoing EDs and DEBs. Finally, our small sample sizes of minorities with EDs or DEBs limited our ability to explore intersectionalities, such as racial/ethnic or gender differences in outcomes. For example, in this study, only 100 non-Hispanic Asians were identified as having ED or DEBs. Future research should explore the similarities and differences in how ED or DEBs influence parenthood for racial/ethnic subgroups and both males and females. In addition, these studies should include additional factors, such as fertility intentions or educational aspirations, which may shape the relationship between ED or DEBs and fertility, and are likely to vary by particular subpopulations.

The current study uses a nationally representative sample of adolescents, allowing for a comparison of those with ED diagnoses or related behaviors to those without such behaviors. This provides a prospective longitudinal cohort type of design and allows for better modeling of potential consequences of ED or DEB. Not to mention, the addition of extensive control variables afforded by a comprehensive survey like Add Health allowed for us to control for life course processes that would not have been possible with clinical studies of more limited scope or covariates.

Conclusion

In conclusion, while existing medical research has commonly stated that EDs result in fertility issues, this study has expanded our understanding of the childbearing

choices of individuals with EDs or DEBs. Most notably, young women who have experienced EDs or DEBs are more likely to have three or more children in early adulthood even when we account for behavioral differences in risk taking and sexual behaviors. EDs in early life may represent a turning point that sets women on a different trajectory of opportunities and constraints in early adulthood compared to women without EDs or DEBs.

Table 4.1 Descriptive Statistics of Estimation Sample

	Females (*N*=5,037)
	% or Mean (*SD*)
Independent Variable	
Diagnosed with ED (Wave III)	
No, never been diagnosed	96.10%
Yes, have been diagnosed	3.90%
Disordered eating behavior (Wave III)	
No DEB	77.66%
Yes DEB	22.34%
ED or DEB (Wave III)	
No	76.52%
Yes	23.48%
Dependent Variable	
Number of children (Wave IV)	
0 kids	43.11%
1-2 kids	44.73%
3+ kids	12.16%
Control Variables	
Race/ethnicity (Wave I)	
Non-Hispanic white	73.75%
Hispanic	6.54%
Non-Hispanic black	15.30%
Non-Hispanic Asian	3.39%
Non-Hispanic other	1.02%
Age (Wave IV) (Range 24-33)	28.13 (1.78)
Sexual orientation (Wave IV)	
Straight	80.32%
Nonstraight	19.68%
Mother's education (Wave I)	
Not sure	3.48%
Less than high school	15.67%
High school GED	35.57%
Some college	19.89%
College degree	18.17%
Post baccalaureate	7.23%
Social support scale (Wave I) (Range 12-40)	32.21 (4.63)
Education (in years) (Wave IV) (Range 8-22)	15.80 (2.33)
Marital status (Wave IV)	
Never been married	42.80%
Married at least once	57.20%
General health (Wave IV) (Range 1-4)	2.67 (0.90)
Depression score (Wave IV) (Range 0-30)	6.39 (4.90)
Body Mass Index (Wave IV) (Range 14.4-80.5)	28.86 (8.12)
Delinquency scale (Wave III) (Range 0-22)	0.55 (1.33)
Age of first vaginal sex (Wave IV) (Range 11-30)	16.78 (2.82)
Number of sexual partners (Wave IV) (Range 1-100)	8.60 (10.01)
Contraceptive use (Wave III)	
None	34.57%
Effective	63.29%
Ineffective	2.14%

Notes: data come from ADD Health. Results are generated based on sample estimation using the longitudinal sample weight (Wave IV) of individuals interviewed at Waves I, III, and IV constructed by ADD Health. ED=Eating disorder; DEB=Disordered eating behavior; *SD*=Standard Deviation

Table 4.2 Unadjusted Empirical Relationship Between ED or DEB and Selected Covariates

	Females (*N*=5,037)	
	ED or DEB (*n*=1,223)	No ED or DEB (*n*=3,814)
	% or Mean (*SD*)	% or Mean (*SD*)
Early Life Context		
Race/ethnicity		
Non-Hispanic white	71.73%	74.39%
Hispanic	7.14%	6.35%
Non-Hispanic black	16.13%	15.03%
Non-Hispanic-Asian	4.05%	3.18%
Non-Hispanic other	1.00%	1.04%
Sexual orientation		
Heterosexual	75.71%***	81.79%***
Any level of same-sex attraction	24.29%***	18.21%***
Mother's education		
Don't know	3.09%**	3.61%**
Less than HS	19.86%**	14.32%**
GED	34.57%**	35.89%**
Some college	17.83%**	20.55%**
College degree	18.87%**	17.94%**
Post baccalaureate	5.78%**	7.69%**
Social support scale	31.55 (4.62)***	32.43 (4.60)***
Health in Early Adulthood		
General health	2.52 (0.95)***	2.71 (0.88)***
Depression score	7.74 (4.61)***	5.96 (4.63)***
Body Mass Index	30.73 (8.58)***	28.26 (7.86)***
Delinquency and Sexual Risk Taking		
Delinquency scale	0.73 (1.39)***	0.50 (1.32)***
Age at first vaginal sex	16.55 (2.71)**	16.86 (2.85)**
Number of sexual partners	10.00 (11.21)***	8.15(9.53)***
Contraceptive use		
None	32.87%	35.12%
Effective	62.44%	62.59%
Ineffective	1.69%	2.29%
Parenthood		
Number of kids		
0 kids	37.85%***	43.75%***
1-2 kids	45.50%***	44.46%***
3+ kids	16.65%***	11.79%***

Notes: Data come from ADD Health; results are generated based on sample estimation using the longitudinal sample weight (Wave IV) of individuals interviewed at Waves I, III, and IV constructed by ADD Health.
Mean differences were assessed using adjusted Wald tests, percent differences were assessed using Pearson Chi-squared tests, comparing women with eating disorders (ED) or disordered eating behaviors (DEB) to those without ED or DEB.
SD=Standard Deviation
*p<0.05 **p<0.01 ***p<0.001

Table 4.3 Multinomial Logistic Regression Results: Relative Risk Ratios

Number of Children	Model 1		Model 2	
	0 kids vs. 1-2	0 kids vs. 3+	0 kids vs. 1-2	0 kids vs. 3+
ED or DEB	1.12	1.48*	1.10	1.46*
	(0.13)	(0.24)	(0.12)	(0.24)
Early Life Context				
Sexual Orientation[a]	0.82	0.81	0.76**	0.68*
	(0.10)	(0.14)	(0.07)	(0.12)
Age (in years)	1.18***	1.23*** (0.05)	1.20***	1.28***
	(0.04)		(0.04)	(0.06)
Race/Ethnicity[b]				
Hispanic	1.14	1.00	1.26	1.19
	(0.23)	(0.30)	(0.23)	(0.34)
Non-Hispanic black	2.62***	5.58***	2.40***	4.91***
	(0.41)	(1.11)	(0.38)	(0.96)
Non-Hispanic Asian	0.86	0.66	1.03	0.86
	(0.21)	(0.23)	(0.25)	(0.33)
Non-Hispanic other	1.47	2.70	1.56	3.00*
	(0.54)	(1.42)	(0.67)	(1.63)
Mom's Education[c]				
Don't know	0.81	0.70	0.90	0.79
	(0.22)	(0.27)	(0.24)	(0.30)
Less than HS	0.95	1.07	0.96	1.09
	(0.18)	(0.23)	(0.17)	(0.22)
Some college	0.81	0.86	0.81	0.85
	(0.10)	(0.17)	(0.10)	(0.16)
College degree	0.65***	0.46***	0.72**	0.53***
	(0.08)	(0.08)	(0.09)	(0.10)
Post Baccalaureate	0.54***	0.22***	0.57**	0.24***
	(0.10)	(0.08)	(0.11)	(0.09)
Social Support Scale	0.98*	0.96**	0.99	0.98
	(0.01)	(0.01)	(0.01)	(0.01)
Early Adult Context				
Education (in years)	0.74***	0.61***	0.77*	0.64***
	(0.02)	(0.02)	(0.09)	(0.03)
Married[d]	4.78***	8.60***	4.58***	8.65***
	(0.50)	(1.36)	(0.53)	(1.33)
Depression score[e]	0.99	0.99	0.99	0.99
	(0.01)	(0.01)	(0.01)	(0.01)
General health[f]	0.91	0.87	0.95	0.91
	(0.05)	(0.08)	(0.04)	(0.08)
Body Mass Index (BMI)	0.99	0.99	0.99	1.00
	(0.01)	(0.01)	(0.01)	(0.01)
Delinquency and Sexual Risk Taking				
Delinquency[g]			0.97	1.00
			(0.03)	(0.05)
Age at first vaginal sex (years)			0.86***	0.77***
			(0.02)	(0.03)
Number of sexual partners			1.00	1.00
			(0.00)	(0.01)
Contraceptive use[h]				
No contraception			1.09	0.96
			(0.12)	(0.15)
Ineffective contraception			0.61	0.77
			(0.20)	(0.39)
N		5,037		5,037
Population size		8,199,076		8,199,076
Strata (region)		4		4

Notes: Data come from ADD health; Estimates use sample weights (Wave IV). Linearized standard errors in parentheses; ED=Eating disorder; DEB=Disordered eating behavior; *p<0.05 **p<0.01 ***p<0.001

[a]compares those who identify as having any level of same-sex attraction to those who identify as 100% heterosexual (reference group)

[b]reference group is non-Hispanic white

[c]reference group is mother with GED or high school diploma

[d]compares those who have never been married to those who have been married at least once (reference group)

[e]higher scores indicate more depressive symptoms

[f]higher scores indicate higher quality of self rated general health

[g]higher scores indicate more delinquent behaviors

[h]reference group is effective contraception

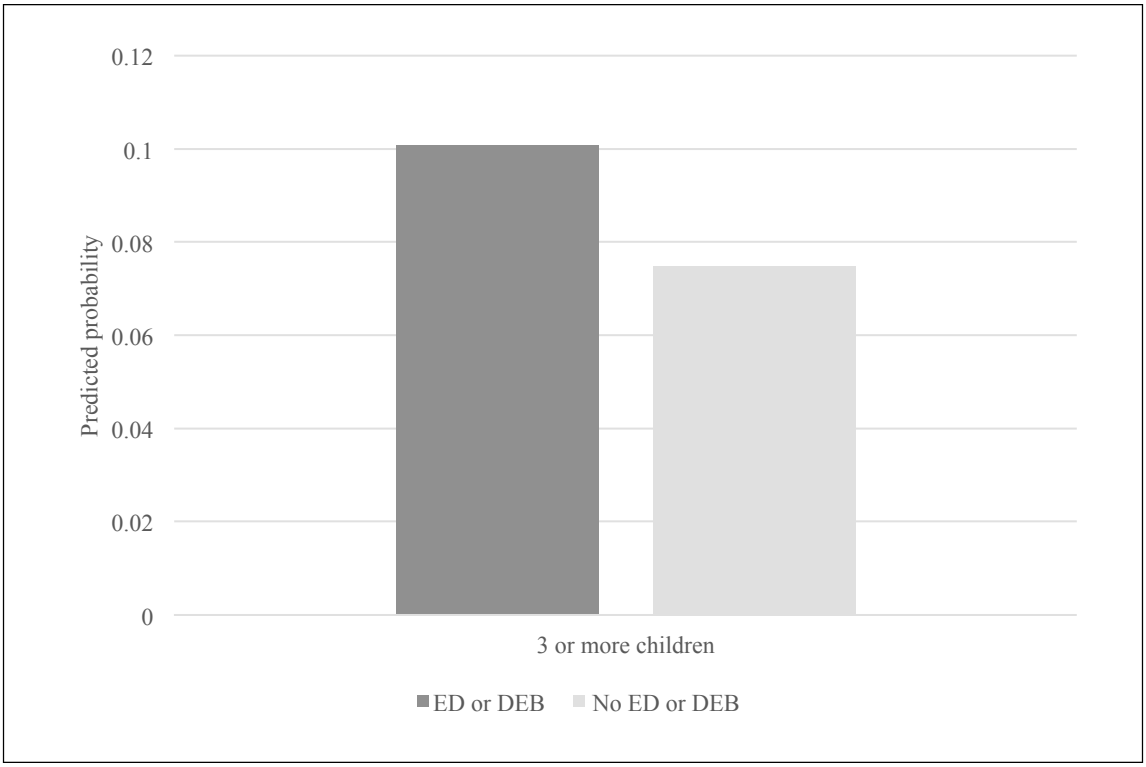

Figure 4.1 ED or DEB and the Probability of Having Three or More Children. Note that the figure depicts effect of eating disorder (ED) or disordered eating behavior (DEB) on the expected change in the probability of having three or more children (all other covariates are held at mean). Results are based on multinomial logistic regression results generated based on sample estimation using the longitudinal sample weight (Wave IV) of individuals interviewed at Waves I, III, and IV constructed by ADD Health. Predicted probability of having three or more children for women with ED or DEB compared to women without ED or DEB is significantly different at $p<0.05$ (using *mgen*, *dxdy*).

CHAPTER 5

CONCLUSIONS AND DIRECTIONS FOR FUTURE RESEARCH

An estimated 20 million American women alive today have or will suffer from an eating disorder (Wade et al. 2011), although many women with eating disorders are unlikely to receive treatment (Hudson et al. 2007). The drive for, or preoccupation with, thinness, the primary motivation of an eating disorder, begins in childhood and evolves across the life course (Collins 1991). Over 40 percent of 1^{st}-3^{rd} -grade girls express a desire to be thinner (Collins 1991), and between 30-50 percent of adolescent girls report engaging in disordered eating behaviors, including crash dieting, fasting, vomiting, taking diet pills, or abusing laxatives to lose weight (Neumark-Sztainer 2005; Tabler and Utz 2015). Both disordered eating behaviors and clinical eating disorders have been found to be associated with adverse physical, mental, and social wellbeing (Berkman et al. 2007; Crow et al. 2009; Nicholls et al. 2011; Norris et al. 2012; Padierna et al. 2000; Striegel-Moore et al. 2003; Swanson et al. 2011; Tabler and Utz 2015).

The study of eating disorders, particular related to their long-term outcomes, has been largely approached from a biomedical perspective. The focus of this dissertation was to bridge epidemiological, psychological, medical, and sociological research on eating disorders in order to better assess whether eating disorders act as a turning point that disrupts successful adult development, in particular, fertility. Fertility is a social behavior that represents an important rite of passage into adulthood: the transition to

parenthood (Buchmann 1989). This study sought to expand our understanding of eating disorders by assessing the long-term consequences of eating disorders on female fertility experiences, using both community and clinical samples. This project had three primary objectives: (1) to assess the influence of eating disorders on fertility trajectories; (2) address the role of familial and shared early life environment on fertility trajectories of women with eating disorders, and assess whether there are differences in fertility trajectories by eating disorder type; and (3) suggest and test potential social mechanisms through which eating disorders may influence fertility. The following paragraphs summarize the contributions made by this project by providing an overview of its results, noting its limitations and providing recommendations for future research.

Review of Study Results

The *first* objective of this study was to assess the influence of eating disorders on fertility trajectories. More specifically, I sought to answer two primary questions: (1) do eating disorders influence fertility timing (age at first birth) and parity (completed fertility)? and (2) does eating disorder measurement and sampling influence results? While prior research indicates that eating disorders may reduce a woman's ability to become pregnant through the physical health consequences of eating disorders on nutrition and bodyweight (Cousins et al. 2015; James 2001; Jokela et al. 2008), very little was known about the overall fertility experiences of women with eating disorders.

In Chapter 2, I compared the age at first birth and parity of women who have been hospitalized or treated for an eating disorder (clinical measurement) in the State of Utah to that of a nationally representative sample of young women who self-identify as having been diagnosed with an eating disorder or engage in disordered eating behaviors

(nonclinical measurement). As outlined by Cohen and Cohen (1984), clinical sampling represents a population that is currently suffering from a disease, while community or population samples tend to represent a population that has ever experienced a disease. Also, a clinical sample is likely to capture the most severe cases of an illness. Chapter 2 extends our understanding of how eating disorders influence fertility by examining eating disorders beyond clinical settings.

Results from these analyses indicate that eating disorders and disordered eating behaviors *do* alter fertility trajectories, but that eating disorder measurement and sampling influences results. In particular, eating disorders were found to *delay* and *reduce* fertility of women of the Utah Population Database (UPDB) sample, which relies on *clinical* measurement, while eating disorders or disordered eating behaviors were found to *advance a*nd *increase* fertility of women within the Longitudinal Study of Adolescent and Young Adult Health (Add Health) sample, which relied on *nonclinical* self-reported measurement of behavior and prior diagnosis. These results are summarized in Figure 5.1.

Chapter 2 results highlight how sampling influences the inferences we make regarding *how* eating disorders influence fertility trajectories. These results should act as a caution to those assuming that clinical (i.e., use of diagnosis) and nonclinical eating disorder measurement (i.e, use of proxy measures such as disordered eating behavior) will yield similar results. In addition, these results indicate that there may be two sets of competing mechanisms that influence fertility among women with eating disorders: social mechanisms that may be associated with increased fertility, and physical or mental health mechanisms that may be associated with reduced or delayed fertility.

The *second* objective of this study was to address the role of familial and shared early life environment on fertility trajectories of women with eating disorders. More specifically, I sought to answer two primary questions: (1) do the fertility experiences of women with eating disorders differ in comparison to their closest aged sister without an eating disorder? and (2) do fertility experiences of women with eating disorders vary by severity or type (i.e., anorexia, bulimia, and eating disorder-not otherwise specified)? In Chapter 3, using data from the Utah Population Database, I compare the age at first birth and parity of women in early adulthood who have been diagnosed with an eating disorder, to that of their closest aged sister who has never been diagnosed with an eating disorder. Although the literature suggests that eating disorders are rooted in familial and shared early life environmental, and/or epigenetic etiology (Campbell et al. 2011), very few studies have actually addressed these potential confounding factors in outcomes research. In addition, I more closely examined how eating disorder type—anorexia nervosa, bulimia nervosa, or eating disorder-not otherwise specified (NOS)—influences fertility trajectories when accounting for measures of disease severity and treatment timing (age at first diagnosis, hospitalization, body mass index).

Chapter 3 extends our understanding of the influence of eating disorders on fertility trajectories in two ways: first, by more robustly examining whether or not the condition *itself*, or the underlying familial or genetic etiology, influence fertility by using a discordant pair, matched sibling design; and second, by examining how eating disorder disease type influences fertility trajectories. As outlined by Donovan and Susser (2011), sibling analyses are particularly useful in testing the robustness of causal relationships that have been identified and confirmed in studies of unrelated individuals. In Chapter 2,

using UPDB data, I confirmed that women with eating disorders (clinical measurement) experienced reduced and delayed fertility. Results from Chapter 3 indicated that while women with anorexia or eating disorder-NOS experienced delayed and reduced fertility in comparison to their sisters, women with bulimia did not. In addition, women with bulimia were found to have increased fertility in comparison to women with anorexia or eating disorder-not otherwise specified (eating disorder-NOS) even when I controlled for treatment timing (age at first diagnosis) and severity (eating disorder hospitalization and body mass index). These results are illustrated in Figure 5.2.

While it is not possible to disentangle which particular set of shared early life experiences may be driving the similarity in fertility between discordant sisters (i.e., genetic factors, familial experiences), results of this project highlight that the sisters of women with eating disorders *do* have more similar fertility experiences than compared to the general population. These results indicate that we may be underestimating how much the etiology of eating disorders, in particular the genetic or familial environmental factors, explain reduced or delayed fertility of women with eating disorders. In addition, this study contributes substantially to our understanding of potential differences in the consequences of particular eating disorder diagnoses by highlighting that women with bulimia have the least altered fertility experiences. In short, this study reconfirms the public health message that all forms of eating disorders may have deleterious consequences for health and wellbeing, but that fertility interventions and screenings may be most beneficial for women with anorexia or eating disorder-NOS.

The *third* objective of this study was to suggest and test potential social mechanisms through which eating disorders may influence fertility. In particular, I sought

to answer why young women with eating disorders or disordered eating behaviors would have high and early fertility, as I saw using the community-based samples in Chapter 2. In Chapter 4, using data from Add Health, I examine more closely adolescent delinquency and sexual risk-taking behavior as potential mediating factors through which eating disorders may be positively associated with higher parity.

Results of this study act as a counterpoint to traditional biomedical literature, which suggests that the physical health consequences of eating disorders result in delayed fertility and fertility complications for women (Bulik et al. 1999; Easter et al. 2011). This study contributes to the literature by illustrating that, in a national sample using nonclinical measurement, eating disorders or disordered eating behaviors are predictive of *increased* fertility in early adulthood, even when accounting for adolescent delinquency and sexual risk taking. This project better assesses two sets of competing mechanisms that influence fertility among women with eating disorders: social mechanisms that co-occur with an ED population that may be associated fertility, and physical or mental health mechanisms that reduce or delay fertility. The conceptual model of Chapter 4 is depicted in Figure 5.3.

In particular, this study indicates that while eating disorders may result in negative health consequences, young women with eating disorders or disordered eating behaviors may be engaging in a constellation of risk-taking behaviors that could potentially increased their risk of early and increased fertility. The association between eating disorders and additional risk-taking behaviors supports adolescent decision-making theory positing that adolescents with reduced self-esteem may be even less likely to appropriately understand or respond to the risk or consequences of, and thus more

likely to engage in, risky behaviors (Smith et al. 1997; Wheeler 2010).

Overall, the goal of this dissertation was to assess whether eating disorders act as a turning point that disrupts successful adult development. Results of this project indicate that eating disorders influence at least one marker of adult development: fertility, albeit through competing social and physiological mechanisms. It is a commonly held view by the public that eating disorders are egocentric, that is, merely an individual's lifestyle choice (Crisp 2005). Given that public sentiment stigmatizes eating disorders and identifies them as a less serious health concern than other mental illnesses such as depression (Roehrig and McLean 2010), we as a society may be underestimating the severity and long-term implications of adolescent eating disorders. This dissertation research indicates that both eating disorders and disordered eating behaviors represent a significant mental illness that acts as a turning point affecting the transition to adulthood. Also, given their influence on fertility, eating disorders or disordered eating behaviors have the potential for intergenerational effects. Children born to younger women or through an unintended pregnancy are more likely to face a host of adverse outcomes, including increased risk of living in poverty and reduced physical and mental wellbeing (Brown and Eisenberg 1995). Eating disorders and disordered eating behaviors potentially influence future generations and have lifelong implications for human development.

Although this research makes substantial contributions to the eating disorder, epidemiological, and sociological literatures, there are several limitations of this project, with potential for additional research. In the following sections, I will discuss the limitations of the current study, and propose directions for future work. I will focus on

three areas of future research in particular: the role of marriage in the association of eating disorders and fertility, whether and how eating disorders and disordered eating behaviors covary with adolescent risk-taking behaviors, and the influence of eating disorders on the adult development of men.

Study Limitations and Future Directions

Study Limitations

First and foremost, the findings reported here are bound by measurement and sampling. In other words, the extent to which such findings apply to different types of samples with different eating disorder or disordered eating behavior measurements is unknown. Results from Chapter 2 confirm that measurement and sampling have implications for the inferences we make about eating disorders. As shown in Figure 5.4, eating disorder research can be organized into four types depending on the use of clinical versus nonclinical sampling, and clinical versus nonclinical measurement. This study relied on two nonclinical samples: a population-based sample (of the state of Utah) from UPDB, and a nationally representative sample from Add Health. A clinical sample may yield significantly different results, as clinical samples typically represent individuals with increased severity or duration, and who are seeking treatment (Cohen and Cohen 1984). Community-based samples are more likely to include those with less serious eating disorder and those who refuse or are unable to seek treatment.

In addition, although this study relied on both clinical and nonclinical measurement (diagnosis within the UPDB sample and self-reported behaviors and experiences in the Add Health sample), our results are bound by the particular eating disorder and disordered eating measures available. For example, although UPDB utilizes

a clinical measure of eating disorders, with three diagnostic categories (anorexia, bulimia, eating disorder-NOS) relating to specific ICD-9 codes, eating disorder diagnostic categories have changed over time, particularly with the separation of binge eating disorder (characterized by excessive eating) from the eating disorder-NOS category within the DSM-5 and use of ICD-10 diagnostic categories (APA 2013). Although research indicates that women with binge eating disorder are more likely to be obese or overweight (White and Gianini 2013), and experience higher rates of miscarriage (Linna et al. 2013), additional research is necessary to understand how binge eating (as a behavior or a diagnosed behavior) may be influencing fertility trajectories.

Another limitation of this project is that it does not take into account fertility intentions. Such information was not available in the data sources used for these analyses. Although discrepancies between stated intent and actual fertility are common (Morgan and Rackin 2010; Quesnel-Vallée and Morgan 2003), differences in fertility experiences may be an artifact of differential desires for parenthood across individual women and families, among women with eating disorders (compared to women without), or by eating disorder type. For example, siblings tend to have more similar fertility experiences than compared to the general population, as was seen in Chapter 3 where discordant sister pairs were found to have more similar fertility experiences than the general population. The similarity in the fertility experiences between siblings may be the result of shared family preferences (Axinn et al. 1994; Lyngstad and Prskawetz 2010).

The literature indicates that women with eating disorders may have lower fertility intentions than women without eating disorders. For example, women with eating disorders are more likely to express unhappiness with being pregnant (Easter et al. 2011),

and are more likely to induce abortion following a pregnancy (Linna et al. 2013). In a qualitative study, women with eating disorders expressed anxiety in early pregnancy due to body changes and weight gain, perceiving these changes as a loss of control over their body or weight (Taborelli et al. 2015). Thus, women with eating disorders may have lower fertility intentions, potentially due to fears of weight gain, which may be translating to lower completed or delayed fertility. This may be particularly true for women with anorexia or bulimia, which are associated with greater fears of weight gain (APA 2013).

However, there may be reasons to anticipate that women with eating disorders may have increased fertility intentions; disadvantaged women more broadly have been found to experience heightened insecurity and fears surrounding whether or not they may be able to achieve motherhood, driving them to pursue it at early ages (Edin and Kefalas 2005). Similarly, becoming pregnant may be more highly valued by women with eating disorders or disordered eating behaviors because they fear they might not have the opportunity achieve a successful pregnancy (or pregnancies) (Martyn-Nemeth et al. 2009). Because this study was unable to account for the fertility intentions of women with eating disorders, it is unclear whether their altered fertility experiences are intentional. Research should therefore assess the fertility intentions of women and adolescents with eating disorders.

Future Research

The limitations of this study are opportunities for future research. In the next section, three particular areas of future research are focused on, and include preliminary findings from the data used in this dissertation (Add Health and UPDB) to justify their

examination: (1) future research should more robustly examine the risk-taking behaviors of adolescents with eating disorders. (2) Future research should examine how eating disorders influence other markers of adult development, including marriage. Finally, (3) future research should explore the later life experiences of men with eating disorders.

Eating Disorders, Adolescent Decision Making, and Risk-taking Behavior

As discussed briefly in Chapter 4, adolescence and the transition to adulthood is often described as a time when individuals engage in risk-taking behaviors (Steinberg and Silverberg 1986), such as disordered eating behaviors (Neumark-Sztainer et al. 2011), substance use (Adams et al. 2002; Chen and Jacobson 2012), or risky sexual behavior (Eaton et al. 2012). Given that adolescents who engage in risky behaviors are more likely to take subsequent, or additional risks—for example, adolescents who engage in substance use are more likely to engage in risky sexual behaviors throughout the transition of adulthood (Tapert et al. 2001)—the presence of eating disorders in adolescence or young adulthood may indicate a constellation of deviant or risk-taking behaviors. However, Chapter 4 has an arguably limited scope, as it focused on only two sets of risk-taking behaviors (delinquency and sexual behaviors), and did not *robustly* assess the relationship between eating disorders and other risk-taking behaviors. Figure 5.5 presents a conceptual model of eating disorders and adolescent risk-taking behaviors that could be further tested.

Focusing on the left-hand side of Figure 5.5, future research should more robustly assess whether eating disorders and other types of risk-taking behaviors are correlated *while* controlling for the etiology of eating disorders or other potential confounding factors that are captured by the individual and family context. Multivariate Analysis of

Covariance (MANCOVA) methodology, which allows for testing of significant differences between group means while controlling for error that may be introduced by covariates (Long and Freese 2006), could be used to examine the association between eating disorders or disordered eating behaviors and a series of additional risk-taking behaviors while controlling for potentially confounding factors.

Future studies could include a range of risk-taking behaviors (e.g., sexual behaviors, delinquent behaviors, health behaviors), to assess which types of risk-taking behaviors are associated with eating disorders. Analyses in Chapter 4 included only two types of behaviors: delinquency and sexual risk-taking. Future research should include other risky health behaviors, such as suicidal/self-injurious behavior and substance use. Prior research indicates that eating disorders are associated with substance use and suicide attempts/self-inflicted injury (Krahn 1991; Piran and Robinson 2011), and that individuals with eating disorders exhibit more impulsive behaviors than their peers (Dawe and Loxton 2004). However, these behaviors are among the most extreme risk behaviors. There may be an even greater correlation among the decisions and attitudes characterized by adolescents who engage in eating disorders and those who engage in other less extreme or more common behaviors, such as sexual behaviors. Given that eating disorders are likely to go undetected, understanding which risk-taking behaviors are most likely to co-occur with eating disorders could help parents and professionals anticipate when an adolescent is at risk of engaging in disordered eating or other risk-taking behaviors.

By adopting this conceptual model of adolescent risk taking (Figure 5.5), future research could better examine how eating disorders and related risk-taking behaviors

might result in cumulative disadvantages in adulthood. Data from the Add Health sample indicate that not only are women with eating disorders or disordered eating behaviors likely to have three or more children in early adulthood, as we saw in Chapter 4, but also that women with eating disorders may be at increased risk of their first child being born out of wedlock; descriptive statistics indicate that of women who have had children, 60.2 percent of women with an eating disorder or disordered eating behavior had a first child while unmarried, and 53.6 percent of women without an eating disorder had a first child while unmarried ($p<0.05$, based on Chi-squared test). Although many women may go on to get married after having a child out of wedlock, single motherhood poses risks to the wellbeing of mothers and children (Avison 1997; Christopher et al. 2002; Graefe and Lichter 1999; Rousou et al. 2013).

These Add Health data should be further explored in order to solidify the life course model used throughout this dissertation to illustrate how eating disorders and disordered eating behaviors exhibited in adolescence may be a turning point that alters the trajectory of adult development. Understanding the constellation of risky behaviors that covary with eating disorder behavior may provide insight on how to identify adolescents who are at risk and potentially intervene during adolescence ensure a more successful transition to adulthood.

Eating Disorders and Marriage

This dissertation focused on one particular marker of adult development: fertility. However, the transition to adulthood is a complex process, involving multiple biological transitions (e.g., physical and sexual development or maturation) and role transitions (e.g., marriage or employment) (Arnett 2000; Arnett 2001). Future research should

examine how eating disorders influence other aspects of adult development. Research that I conducted prior to this dissertation project indicates that eating disorders and disordered eating behaviors may be disruptive to markers of socioeconomic independence, such as personal income, home ownership, and levels of education (Tabler and Utz 2015); however, few if any studies have assessed how eating disorders shape marriage trajectories. Marriage is an important marker of adulthood (Arnett 2001), and is likely associated with the fertility behaviors assessed here. Future research should examine how eating disorders influence marriage experiences, and whether marriage trajectories provide additional context for understanding the association between disordered eating behavior and fertility.

The social selection hypothesis posits that better adjusted, healthier individuals become and remained married (Blane, Smith, and Bartley 1993). Research has confirmed the social selection hypothesis, indicating that healthier individuals have more positive marital experiences than those in poor health (Horn et al. 2013; Stutzer and Frey 2006). Building on this prior research, future research could assess the influence of eating disorders on marriage behavior. Based on the social selection hypothesis, eating disorders should, hypothetically, negatively select one for marriage.

Both UPDB and Add Health data contain markers of marital timing and marital experiences. Future research could replicate the study design of Chapter 2, which compared the fertility trajectories of individuals with eating disorders or disordered eating behaviors within the Add Health and UPDB samples. However, instead of examining fertility trajectories, a future study could examine marriage trajectories, including marital timing and marital transitions such as divorce and remarriage.

Preliminary descriptive statistics of marriage experiences of these two samples are presented in Table 5.1. Results presented in Table 5.1 indicate that there are differences in the marital experiences of women with eating disorders and those without. For example, both women with eating disorders in the Add Health sample and UPDB sample report higher rates of divorce compared to their unafflicted peers. Therefore, it is possible for a future study to robustly examine the relationship between eating disorders and marriage trajectories.

Assessing the relationship between eating disorders and marriage is important not only because marriage behaviors influence social wellbeing, but also because successful marriage is known to have beneficial mental and physical health outcomes (Horn et al. 2013; Wade and Pevalin 2004). In addition, understanding marriage experiences could provide new insight into the association between eating disorders and fertility. Marital transitions are some of the most important life experiences that influence both fertility intentions and behaviors (Schoen et al. 1999). Understanding the adult development, including marriage experiences, of individuals with eating disorders would provide insight into how eating disorders influence markers of physical, mental, and social wellbeing across the life course.

Eating Disorders among Men

This project, like the vast majority of research on eating disorders and disordered eating behaviors, only examined the experiences of women. The focus of the eating disorder literature on females is due to the fact that women have been consistently shown to have higher lifetime prevalence of eating disorders than males, and are more likely to engage in disordered eating behaviors (Hudson et al. 2007; Santos et al. 2007; Stephen et

al. 2014). Because the current project focused exclusively on fertility-related outcomes, a female focus was appropriate here. However, there is a growing literature on eating disorders and disordered eating behaviors among men. Many studies indicate clinical similarities between males and females with eating disorders (Woodside et al. 2001), and that males and females have similar patterns of age at onset (Woodside et al. 2001), but different symptomology (Lewinsohn et al. 2002). Previous studies also suggest that the etiological factors influencing eating disorders or disordered eating behavior development is similar for both males and females (Burns and Crisp 1985; Polivy and Herman 2002; Woodside and Kaplan 1994).

While the etiology, and physical and mental health consequences are similar for males and females, it is important to not assume that eating disorders will have similar influences on social wellbeing for men and women. For social outcomes in particular, it is important to keep in mind that "gender" is conceptually different from "sex" or a "sex category" (West and Zimmerman 1987). Although the physical manifestation and physical outcomes related to eating disorders may be similar for men and women (which many in large be because these studies are engaging in a comparison of sex or sex category differences), it is important to keep in mind that gender is a *social* construct, and therefore, *social* outcomes may vary by gender. When men and women appear to function similarly in relation to a given phenomenon, we sometimes have the tendency to minimize differences, that is, engage in "beta bias," where the similarity or equality between men and women may become over stressed (Hare-Mustin and Marecek 1988). It is important to consider our own potential to engage in "beta bias" by bringing greater attention in the future to the male experience of eating disorders.

Both UPDB and Add Health data contain subsamples of males with eating disorders or disordered eating behaviors. Preliminary analyses indicate that within Add Health 708 males self-report having been diagnosed with an eating disorder or having a disordered eating behavior (out of 5,934 males total), and that UPDB contains 991 males diagnosed with an eating disorder (out of 3,931 males total). In a previous study, I completed prior to this dissertation work using Add Health data, I found that while an eating disorder or disordered eating behavior was associated with reduced socioeconomic attainment in early adulthood for women, adolescent eating disorder or disordered eating behavior was not associated with the socioeconomic attainment of men (Tabler and Utz, 2015). Future research should examine the fertility and other adulthood experiences of men with eating disorders.

Considering potential gender differences in the influence of eating disorders on adulthood development is important given that the transition to adulthood *itself* varies by gender. In general, females are at risk of lower socioeconomic attainment (Hegewisch et al. 2010), younger ages of first birth (Martinez, Daniels, and Chandra 2006), and earlier entry into marriage than males (Schoen and Standish 2001). Women with lower levels of educational achievement have earlier and higher fertility (Dye 2008; Monte and Ellis 2009). Similar trends exist for men, although the educational gradient is less steep (Martinez et al. 2006). It is therefore important to consider potential gender differences when assessing the relationship between eating disorders and markers of adult development, particularly fertility and marriage experiences.

Conclusion

The focus of this dissertation was to bridge epidemiological, psychological, medical, and sociological research on eating disorders in order to better assess whether eating disorders act as a turning point that disrupts successful adult development, in particular, fertility. This study sought to expand our understanding of eating disorders by assessing the long-term consequences of eating disorders and disordered eating behaviors on female fertility experiences, using both community and clinical samples. This project had three primary objectives: (1) to assess the influence of eating disorders on fertility trajectories; (2) address the role of familial and shared early life environment on fertility trajectories of women with eating disorders, and assess whether there are differences in fertility trajectories by eating disorder type; and (3) suggest and test potential social mechanisms through which eating disorders may influence fertility. Future research could expand on this dissertation by more robustly examining the association between risk-taking behaviors of adolescents and disordered eating behaviors, examining how eating disorders influence other markers of adult development, including marriage, and by exploring the adulthood experiences of men with eating disorders.

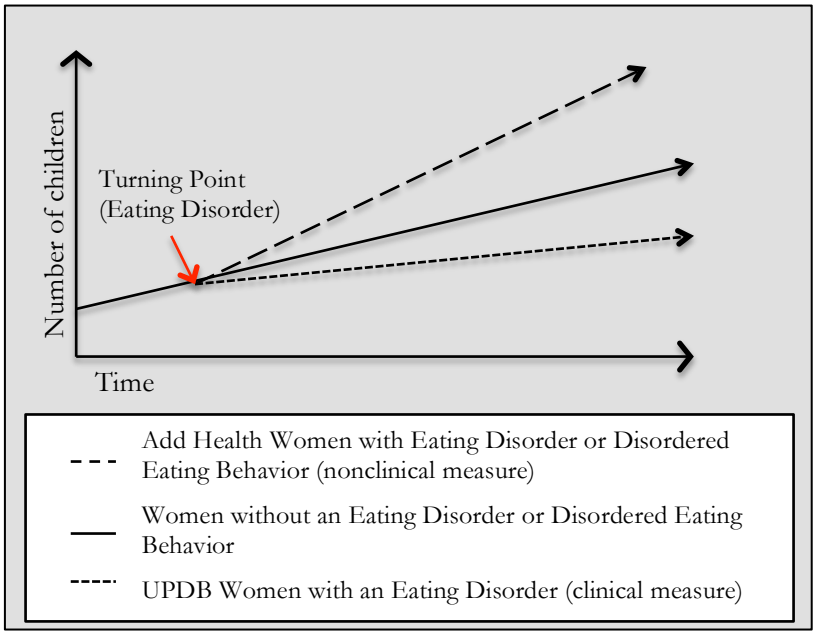

Figure 5.1 Influence of Eating Disorders on Fertility Trajectories of Two Samples

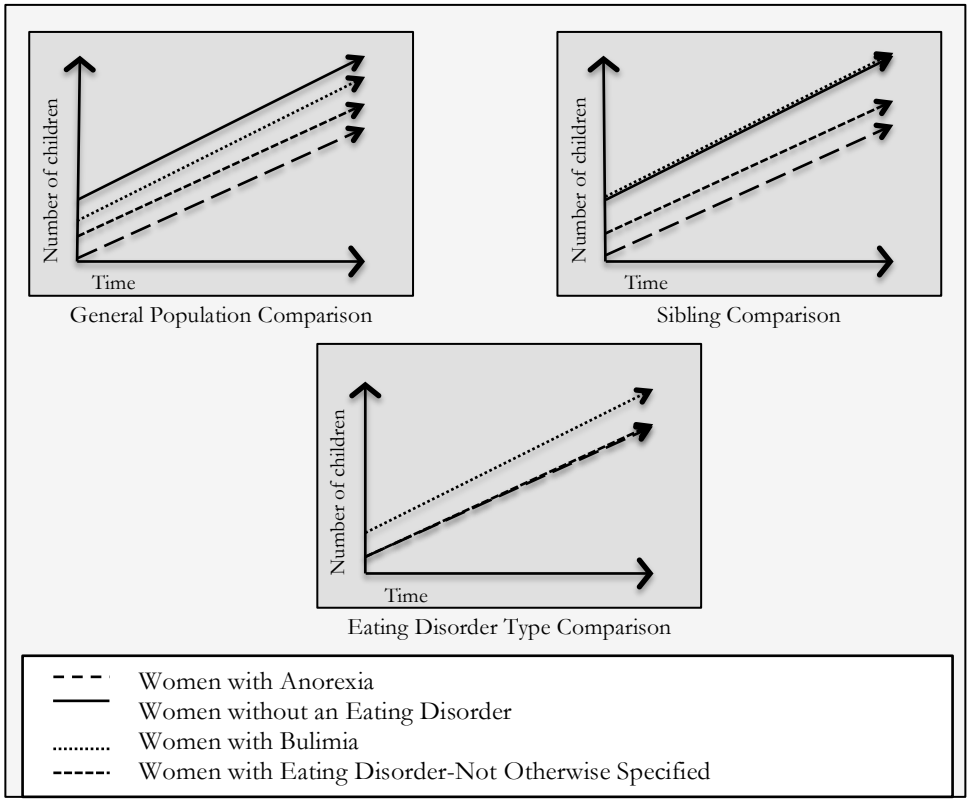

Figure 5.2 Influence of Eating Disorder Type on Fertility Trajectories

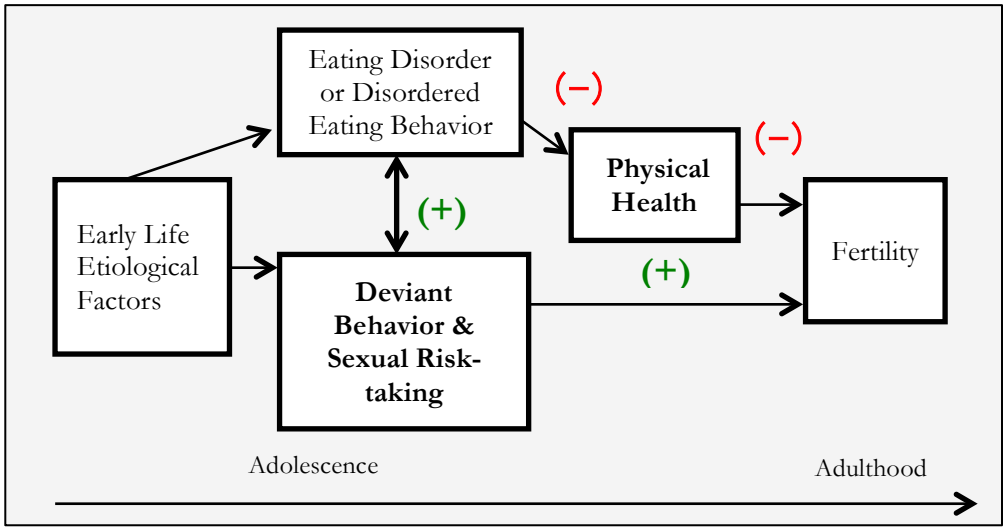

Figure 5.3 Eating Disorder or Disordered Eating Behavior and Fertility: Competing Mechanisms. Notes that - denotes a hypothesized negative association while + denotes a hypothesized positive association.

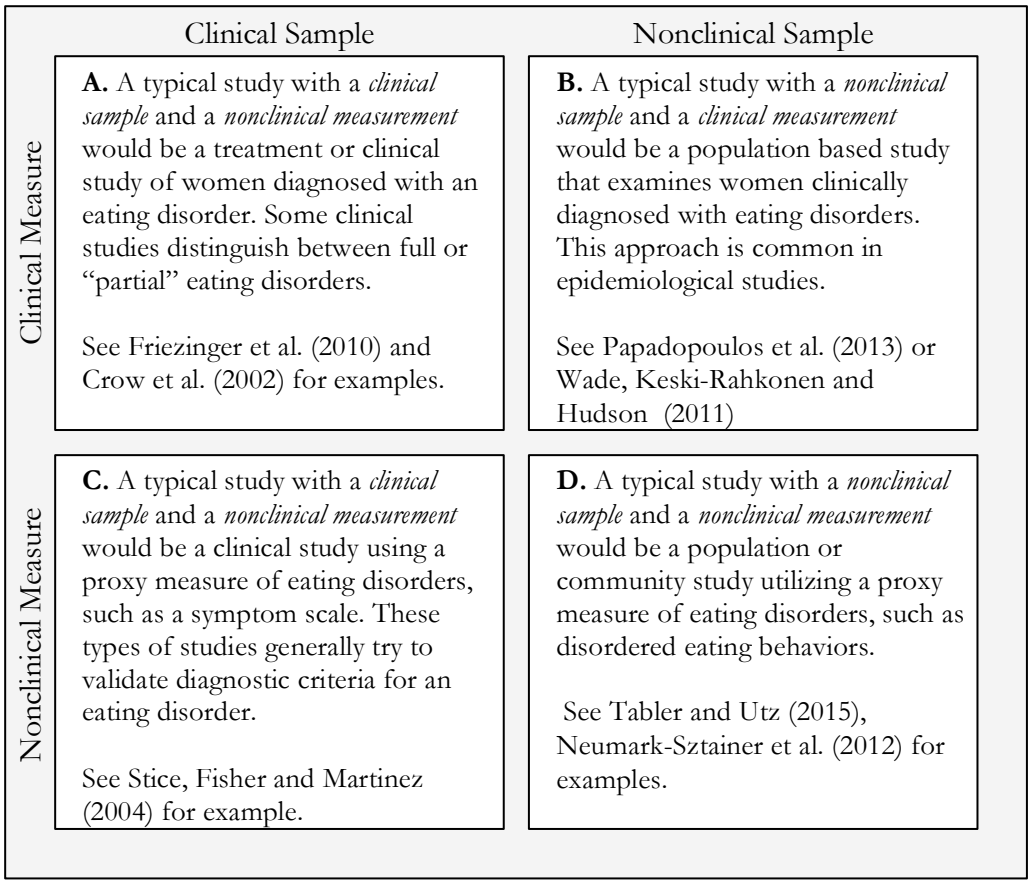

Figure 5.4 Clinical versus Nonclinical Sampling and Measurement

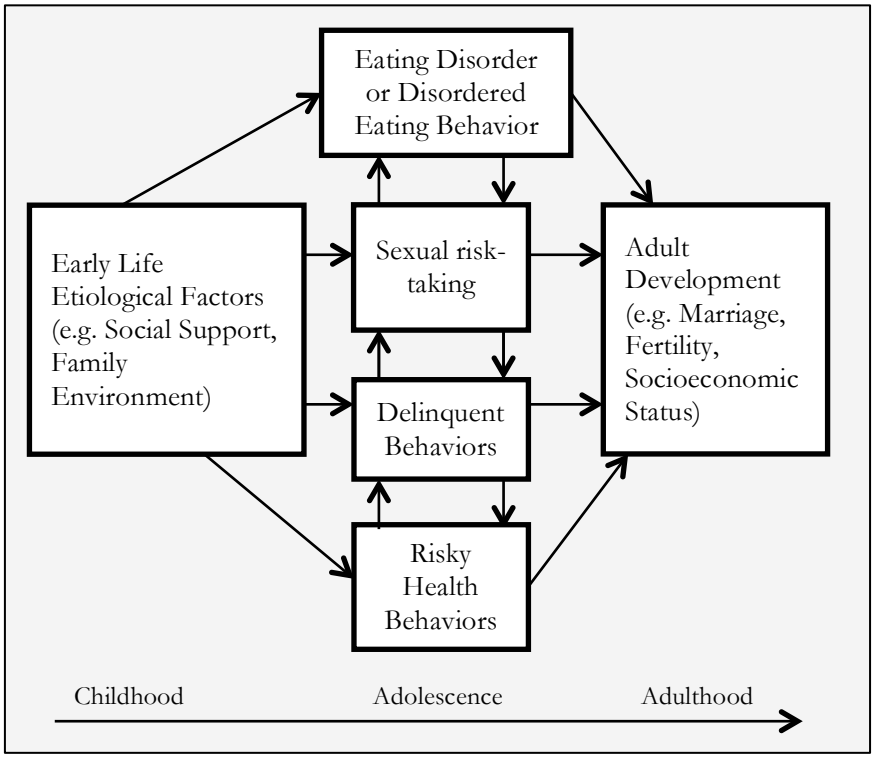

Figure 5.5 Eating Disorder or Disordered Eating Behavior and Adolescent Decision Making

Table 5.1 Unadjusted Empirical Relationship Between ED or DEB and Marriage Experiences

	Add Health females (*N*=5,343)		UPDB females (*N*=22,970)	
	ED or DEB (*n*=1,255)	No ED or DEB (*n*=4,088)	ED (*n*=5,761)	No ED (*n*=17,209)
	% or Mean (*SD*)	% or Mean (*SD*)	% or Mean (*SD*)	% or Mean (*SD*)
Married once	54.82%*	54.51%*	36.68%***	44.30%***
Ever been divorced	15.14***	10.69%***	16.72%***	11.69%***
Ever been remarried	5.82%**	3.91%**	7.34%***	4.98%***
Age at first marriage	22.67(3.22)*	22.98(3.16)*	23.34(5.84)***	22.85(5.48)***

Notes: Data come from ADD Health and UPDB.
Mean differences were assessed using two tailed *t*-tests, percent differences were assessed using Pearson Chi-squared tests, comparing women with eating disorders (ED) or disordered eating behaviors (DEB) to those without ED or DEB.
SD=Standard Deviation
*$p<0.05$ **$p<0.01$ ***$p<0.001$

APPENDIX

ADD HEALTH SCALES AND MEASURES

Wave I

Protective Environment Scale (reliability coefficient=0.78)

Responses include: 1 “Not at all” 2 “Very Little” 3 “Somewhat” 4 “Quite a Bit” 5 “Very Much”

Item 1: “How much do you feel that adults care about you?”

Item 2: “How much do you feel that your teachers care about you?”

Item 3: “How much do you feel that your parents care about you?”

Item 4: “How much do you feel that your friends care about you?”

Item 5: “How much do you feel that your people in your family understand you?”

Item 6: “How much do you feel that you want to leave home?” (Reverse coded)

Item 7: “How much do you feel that you and your family have fun together?”

Item 8: “How much do you feel like your family pays attention to you?”

Wave III

Delinquency Scale (reliability coefficient=0.70)

Responses include: 0 “never” 1 “one or two times” 2 “three or four times” and 3 “five or more times.”

Item 1: “In the past 12 months, how often did you deliberately damage property that didn’t belong to you?”

Item 2: “In the past 12 months, how often did you steal something worth more than $50?”

Item 3: “In the past 12 months, how often did you go into a house or building to steal something?”

Item 4: “In the past 12 months, how often did you use or threaten to use a

weapon to get something from someone?"

Item 5: "In the past 12 months, how often did you sell marijuana or other drugs?"

Item 6: "In the past 12 months, how often did you steal something worth less than $50?"

Item 7: "In the past 12 months, how often did you take part in a physical fight where a group of your friends was against another group?"

Item 8: "In the past 12 months, how often did you buy, sell, or hold stolen property?"

Item 9: "In the past 12 months, how often did you use someone else's credit card, bankcard, or automatic teller card without their permission or knowledge?"

Item 10: "In the past 12 months, how often did you deliberately write a bad check?"

Item 11: "In the past 12 months, how often did you use a weapon in a fight?"

Item 12: "In the past 12 months, how often did you carry a handgun at school or work?"

Eating Disorder and Disordered Eating Behavior Measures

Eating Disorder Diagnosis

"Have you ever been told by a doctor that you have an eating disorder, such as anorexia nervosa or bulimia?" *Y/N*

Disordered Eating Behavior

Weight-Related Compensatory Behaviors

"Which of the following things did you do during the past seven days in order to lose weight or stay the same weight? Indicate all that apply."

Item 1: "Fasted or skipped meals."

Item 2: "Made yourself throw up."

Item 3: "Weight loss pills."

Item 4: "Took laxatives."

Item 5: "Used diuretics—that is, water pills."

Binge Eating Behavior

"In the past seven days, have you eaten so much in a short period that you would have been embarrassed if others had see you do it?" *Y/N*

Wave IV

CES-D Index of Depression (reliability coefficient=0.83)

Responses include: 0 "never or rarely" 1 "sometimes" 2 "a lot of the time" 3 "most of the time or all of the time"

"Now, think about the past seven days. How often was each of the following things true during the past seven days?"

Item 1: "You were bothered by things that usually don't bother you."

Item 2: "You could not shake off the blues, even with help from your family and your friends, during the past seven days."

Item 3: "You felt just as good as other people, during the past seven days." (reverse coded)

Item 4: “You had trouble keeping your mind on what you were doing, during the past seven days.”

Item 5: “You felt depressed, during the last seven days.”

Item 6: “You felt that you were too tired to do things, during the past seven days.”

Item 7: “You felt happy, during the past seven days.” (reverse coded)

Item 8: “You enjoyed life, during the past seven days.” (reverse coded)

Item 9: “You felt sad, during the past seven days.”

Item 10: “You felt that people disliked you, during the past seven days”

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